

## WEST Search History

DATE: Friday, November 27, 2003

<u>Set Name</u> side by side	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u> result set
<i>DB: USPT,PGPB,JPAB,EPAB,DWPI; PLUR YES; OP ADJ</i>			
L14	L2 and L12	2	L14
L13	L1 and L12	0	L13
L12	GGGACTTTCC	64	L12
L11	L2 and L9	184	L11
L10	L1 and L9	101	L10
L9	binding sites	37632	L9
L8	L6 and L2	0	L8
L7	L6 and L1	1	L7
L6	L3 and L5	64	L6
L5	dendritic cell	3007	L5
L4	ribozym\$3	8755	L4
L3	tolerogen\$3	395	L3
L2	nf kappa b	424	L2
L1	nuclear factor kappa b	211	L1

END OF SEARCH HISTORY



TABLE 10.1	TABLE 10.2	TABLE 10.3	TABLE 10.4
$\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$ $\frac{1}{2} \times \frac{1}{4} = \frac{1}{8}$ $\frac{1}{4} \times \frac{1}{4} = \frac{1}{16}$	$\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$ $\frac{1}{2} \times \frac{1}{4} = \frac{1}{8}$ $\frac{1}{4} \times \frac{1}{4} = \frac{1}{16}$	$\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$ $\frac{1}{2} \times \frac{1}{4} = \frac{1}{8}$ $\frac{1}{4} \times \frac{1}{4} = \frac{1}{16}$	$\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$ $\frac{1}{2} \times \frac{1}{4} = \frac{1}{8}$ $\frac{1}{4} \times \frac{1}{4} = \frac{1}{16}$

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US 2001-844915 20010427

PRIORITY AFFIN. INFO.: US 2000-200479P P 20000428

AB The present invention relates to a tolerogenic mammalian **dendritic cells** (DCs) and methods for the produ. of the tolerogenic DCs. In addn., the present invention provides a method for enhancing tolerogenicity in a host comprising administering the tolerogenic mammalian DCs of the present invention to the host. The tolerogenic DCs of the present invention comprise an oligodeoxynucleotide (ODN) which has one or more NF- $\kappa$ B **binding sites**. The tolerogenic DCs of the present invention may further comprise a viral vector, and preferably an adenoviral vector, which does not affect the tolerogenicity of the tolerogenic DCs when present in situ. Enhanced tolerogenicity in a host is useful for prolonging foreign graft survival and for treating inflammatory related diseases, such as autoimmune diseases.

L34 ANSWER 3 OF 4 (PAGES 1-11) (PAGES 1-11)

ACCESSION NUMBER: 2001-04-27

DOCUMENT NUMBER: 2001-04-27

TITLE: **Enhancement of cardiac allograft survival using dendritic cells treated with**

**NF- $\kappa$ B decoy oligodeoxynucleotides**  
Giannoukakis, Nick; Bonham, C. Andrew; Qian, Shiguang; Zhou, Zhongyou; Peng, Liansha; Harnaha, Jo; Li, Wei; Thomson, Angus W.; Fung, John J.; Robbins, Paul D.; Lu, Lina

CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA  
SOURCE: Molecular Therapy (2000), 1(5), Pt. 1, 430-437

CODEN: MTHCKK; ISSN: 1525-0016

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB **Dendritic cells** (DC) classically present immune responses for naive and memory T cells and are essential for the development of both Th1 and Th2 responses. DCs are also involved in the regulation of T cell responses. DCs are characterized by their ability to present antigens to T cells. The expression of these cells is altered with NF- $\kappa$ B-dependent transcriptional regulation of their genes. DCs are also involved in the regulation of NF- $\kappa$ B-dependent transcriptional regulation of their genes as well as NF- $\kappa$ B translocation to the nucleus. In this report, we demonstrate that double-stranded oligodeoxynucleotides (ODNs) **binding sites** for NF- $\kappa$ B NF- $\kappa$ B ODN are efficiently incorporated by bone marrow-derived DCs and specifically inhibit NF- $\kappa$ B-dependent transcription of a reporter gene. Moreover, exposure of DC to the oligodeoxynucleotide inhibited lipopolysaccharide (LPS)-induced nitric oxide produ., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF- $\kappa$ B ODN selectively suppresses the cell-surface expression of costimulatory molecules and interferes with MHC class I or class II expression. Furthermore, NF- $\kappa$ B ODN-induced alterations in gene expression are not observed in DCs that are cultured in the presence of LPS. Finally, treatment of NF- $\kappa$ B ODN-treated DCs with LPS results in a significant reduction in the ability of these cells to stimulate T cell responses.

significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-kappa.B and other transcriptional pathways involved in immune stimulation in DC using GDN decoy approaches could be one means to promote tolerance induction in organ transplantation. (c) 2000 Academic Press.

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS REFERENCE. ALL CITED ARE AVAILABLE IN THE REF FORMAT

134 JOURNAL OF IMMUNOLOGY 160(3) 1224-1232

ACCESSION NUMBER: 1999010100000000000000000000000000

DOCUMENT NUMBER: 1999010100000000000000000000000000

TITLE: VEGF and the Flt-1 receptor inhibit NF-kappa.B activation in hemopoietic progenitor cells

dendritic cell maturation through the inhibition of nuclear factor-

kappa.B activation in hemopoietic progenitor cells

AUTHOR(S): Oyama, Tsunehiro; Ran, Sophia; Ishida, Tadao; Nadaf, Sarena; Kerr, Lawrence; Carbone, David P.; Gribilovich, Dmitry I.

CORPORATE SOURCE: The Vanderbilt Cancer Center and Departments of Medicine and Microbiology and Immunology, Vanderbilt University School of Medicine, Nashville, TN, 37232, USA

SOURCE: Journal of Immunology (1998), 160(3), 1224-1232

CODEN: JOIMA3; ISSN: 0022-1767

PUBLISHER: American Association of Immunologists

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vascular endothelial growth factor (VEGF), produced by almost all tumor cells, attracts the ability of hemopoietic progenitor cells (HPC) to differentiate into functional dendritic cells (DC). During the early stages of tumor maturation, in this study we demonstrate specific binding of VEGF to Flt-1. This binding was efficiently competed by pretreated cells with VEGF, a ligand reportedly specific for the Flt-1 receptor. The number of binding sites for VEGF decreased during differentiation in vitro, associated with decreased levels of mRNA for Flt-1. VEGF significantly inhibited nuclear factor-

kappa.B (NF-kappa.B)-dependent activation of reporter gene transcription during the first 24 h in culture. The presence of VEGF significantly decreased the specific DNA binding of NF-kappa.B as early as 30 min after induction with TNF-alpha. This was followed on days 7 to 10 by decreases in the mRNA for RelB and c-Rel, two subunits of NF-kappa.B. Blockade of NF-kappa.B activity in HPC at early stages of differentiation with an adenovirus expressing a dominant I-kappa.B inhibitor of NF-kappa.B reproduced the pattern of expression of VEGF. Thus, NF-kappa.B plays an important role in regulation of HPC to DC, and VEGF activation of the Flt-1 receptor leads to inhibition of the activation of NF-kappa.B in this system. Blockade of NF-kappa.B activation in HPC by tumor-derived factors may therefore be a mechanism by which tumor cells directly suppress the ability of the immune system to respond to the tumor and to suppress tumor growth.

134 JOURNAL OF IMMUNOLOGY 160(3) 1224-1232

ACCESSION NUMBER: 1999010100000000000000000000000000

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TITLE: VEGF and the Flt-1 receptor inhibit NF-kappa.B activation in hemopoietic progenitor cells

AUTHOR(S): Oyama, Tsunehiro; Carbone, David P.; Ishida, Tadao; Kerr, Lawrence; Nadaf, Sarena; Ran, Sophia; Gribilovich, Dmitry I.

CORPORATE SOURCE: Department of Experimental Medicine, University of Medicine, Nashville, TN, 37232, USA

SOURCE: Journal of Immunology (1998), 160(3), 1224-1232

ENGLISH  
DOCUMENT TYPE:  
LANGUAGE:

AB The authors studied the expression of an IL-12 receptor by fresh **dendritic cells** (DC) and T cells. Using RT-PCR, RNase protection, and electrophoretic mobility shift assay anal., they found that DC possess an IL-12 receptor with  $\beta$  subunit (downstream box 1)-related differences from that on T cells. IL-12 signaling through this receptor involved members of the NF- $\kappa$ B but not STAT family. The unique properties of the IL-12 receptor on DC, characterized by a single class of **binding sites** with a  $K_d$  of about 300 pM, may underlie rather unique effects, such as IFN- $\gamma$ -independent augmentation of class II antigen expression and priming for IFN- $\gamma$ -induced prodn. of IL-12.

REFERENCE COUNT: 47 THERE ARE 47 OTHER REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

134 ANSWER 6 OF 4  
ACCESSION NUMBER: 134  
DOCUMENT NUMBER: 134  
TITLE: Regulation of Interleukin-12 p40 transcript by CD40 stimulation via activation of **nuclear factor- $\kappa$ B**  
AUTHOR (S): Yoshimura, Takayuki; Nagase, Hisashi; Ishida, Takaomi; Inoue, Junichiro; Nishuchi, Hideo  
CORPORATE SOURCE: Department Allergy, Institute Medical Science, University Tokyo, Tokyo, 108, Japan  
SOURCE: European Journal of Immunology (1997), 27(12), 3461-3471  
OPEN: EMBASE; ISSN: 0014-2969  
PUBLISHER: Wiley-VCH Verlag GmbH  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Interleukin-12 is produced in response to infection with bacteria or parasites or to bacterial constituents such as lipopolysaccharide (LPS) in monocytes/macrophages and **dendritic cells**, and is generated by the interaction between antigen-presenting cells and antigen-presenting cells via CD40-CD40 ligand (CD40L). Transcriptional analysis of p40 gene revealed that transcriptional enhancers such as LPS response element (LPSRE) and CD40L response element (CD40LRE) were characterized by a NF- $\kappa$ B binding site, NF- $\kappa$ B, and a nuclear factor- $\kappa$ B binding site (NF- $\kappa$ B). These cells, stimulated by an antigen-presenting cell, produced IL-12 by transfection with a CD40L expression vector, pCD40L, which was enhanced p40 mRNA expression. Sequence analysis of p40 promoter identified potential nuclear factor- $\kappa$ B binding sites conserved between mouse and human. Electrophoretic mobility shift assay revealed that the potential NF- $\kappa$ B binding sequence which is located around 120 bp upstream of the transcription initiation site in murine and human p40 genes formed an NF- $\kappa$ B complex with nuclear ext. from Daudi cells stimulated by CD40L ligand. Moreover, transfection of Daudi cells with the polyom. NF- $\kappa$ B binding sequence fused to a thymidine kinase/chloramphenicol acetyltransferase (TK) reporter plasmid greatly induced CAT activity, but transfection with the polyom. mutant NF- $\kappa$ B binding sequence did not. These results suggest that the NF- $\kappa$ B binding site is a major enhancer for p40 gene expression. Transcription induction of the p40 gene by CD40L is dependent on the binding of NF- $\kappa$ B.

136 ANSWER 1 OF 7 MARION WILKINSON 11.1.00

ACCESSION NUMBER: 2002:V12316 CAPLUS

TITLE: Marked prolongation of cardiac allograft survival by **dendritic cells** genetically engineered with NF- $\kappa$ .

AUTHOR(S): B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig.

CORPORATE SOURCE: Department of Immunology and Thomas H. Starzl Transplantation Institute, University of Pittsburgh Medical Center, University of Pittsburgh, Pittsburgh, PA, 15261, USA

SOURCE: Journal of Immunology (2002), 169(6), 3382-3391

ISSN: 0022-1767

PUBLISHER: American Association of Immunologists

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Bone marrow-derived **dendritic cells** (DCs) can be genetically engineered using adenoviral (Ad) vectors to express immunosuppressive mols. that promote T cell unresponsiveness. The success of these DCs for therapy of allograft rejection has been limited in part by the potential of the adenovirus to promote DC maturation and the inherent ability of the DC to undergo maturation following in vivo administration. DC maturation occurs via NF- $\kappa$ .

B-dependent mechanisms, which can be blocked by double-stranded "decoy" oligodeoxynucleotides (ODNs) that bind

sites to NF- $\kappa$ .B. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig.

decoy" oligodeoxynucleotides (ODNs) that bind NF- $\kappa$ .B

decoy" oligodeoxynucleotides (ODNs) that bind NF- $\kappa$ .B. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig.

Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL/10; B6.H-2b) before transplant significantly prolongs MHC-mismatched (Pdx1<sup>0</sup>; C57BL/10) vascularized heart allograft survival, with long-term (100 days) non-rejection graft survival in 40% of recipients. The mechanisms responsible for DC tolerance, which may involve activation-induced apoptosis of all-reactive T cells, do not lead to skewing of intragraft cytokine responses. Use of NF- $\kappa$ .

**kappa.B** antisense decoys in conjunction with rAd encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection or autoimmune disease with minimal or no systemic immunosuppression.

REFERENCE COUNT: 100 THERE ARE 100 CITATION REFERENCES AVAILABLE FOR THIS REFERENCE. ALL CITATIONS AVAILABLE IN THE REFERENCE

136 ANSWER 1 OF 7 MARION WILKINSON 11.1.00

ACCESSION NUMBER: 2002:V12316 CAPLUS

TITLE: Marked prolongation of cardiac allograft survival by **dendritic cells** genetically engineered with NF- $\kappa$ .

AUTHOR(S): B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig. B. olicode oxylid nucleotide (CpG) and adenoviral vector (Ad) encoding CTLA4-Ig.

CORPORATE SOURCE: Department of Immunology and Thomas H. Starzl Transplantation Institute, University of Pittsburgh Medical Center, University of Pittsburgh, Pittsburgh, PA, 15261, USA

SOURCE: Journal of Immunology (2002), 169(6), 3382-3391

ISSN: 0022-1767

PUBLISHER: American Association of Immunologists

dendritic cells

RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

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2001:810672 CARLUS

33 : 355016

The use of tolerogenic dendritic

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1. Definition: A function  $f: X \rightarrow Y$  is called a linear map if it satisfies the following two properties:

1. *Chlorophyll a* (Chl *a*) and *Chlorophyll b* (Chl *b*) were determined by the method of Lichtenthal and Whistler (1973). The total chlorophyll content was determined by the method of Arar and Cook (1980). The carotenoid content was determined by the method of Lichtenthal and Whistler (1973). The total carotenoid content was determined by the method of Arar and Cook (1980). The total protein content was determined by the method of Lowry et al. (1951). The total lipid content was determined by the method of Bligh and Dyer (1959). The total carbohydrate content was determined by the method of Dubois and Gilles (1950). The total nucleic acid content was determined by the method of Burton (1956). The total ash content was determined by the method of AOAC (1990). The total moisture content was determined by the method of AOAC (1990). The total dry matter content was determined by the method of AOAC (1990). The total organic acid content was determined by the method of AOAC (1990). The total alkaloid content was determined by the method of AOAC (1990). The total flavonoid content was determined by the method of AOAC (1990). The total phenol content was determined by the method of AOAC (1990). The total tannin content was determined by the method of AOAC (1990). The total saponin content was determined by the method of AOAC (1990). The total sterol content was determined by the method of AOAC (1990). The total glycoside content was determined by the method of AOAC (1990). The total alkaloid content was determined by the method of AOAC (1990). The total flavonoid content was determined by the method of AOAC (1990). The total phenol content was determined by the method of AOAC (1990). The total tannin content was determined by the method of AOAC (1990). The total saponin content was determined by the method of AOAC (1990). The total sterol content was determined by the method of AOAC (1990). The total glycoside content was determined by the method of AOAC (1990).

1. *Journal of the American Medical Association*, 1997; 278: 1039-1044.

1. The first group of variables includes the demographic characteristics of the respondents, such as age, gender, and education level. These variables are used to control for potential confounding factors that may influence the relationship between the independent and dependent variables.

[illegible]

DEPARTMENT NO.

DATE:

Wang, Y., & Wang, J. (2017). The effect of the 2016-2017 influenza season on the 2018-2019 season in China. *Vaccine*, 35(1), 1-10. doi:10.1016/j.vaccine.2016.11.040.

1. *Journal of the American Medical Association*, 277, 1996, 1033-1037.

W:	AE	AF	AG	AH	AI	AL	AM	AN	AO	AP	AR	AS	AT	AW	AX	AY	BA	BB	BC	BD	BE	BF	BG	BH	BI	BJ	BK	BL	BM	BN	BO	BP	BQ	BR	BS	BT	BU	BV	BW	BX	BY	BZ	CA	CB	CC	CD	CE	CF	CG	CH	CI	CJ	CK	CL	CM	CN	CO	CP	CQ	CR	CS	CT	CU	CV	CW	CX	CY	CZ	DA	DB	DC	DD	DE	DF	DG	DH	DI	DJ	DK	DL	DM	DN	DO	DP	DQ	DR	DS	DT	DU	DV	DW	DX	DY	DZ	EA	EB	EC	ED	EE	EF	EG	EH	EI	EJ	EK	EL	EM	EN	EO	EP	EQ	ER	ES	ET	EU	EV	EW	EX	EY	EZ	FA	FB	FC	FD	FE	FF	FG	FH	FI	FJ	FK	FL	FM	FN	FO	FP	FQ	FR	FS	FT	FU	FV	FW	FX	FY	FZ	GA	GB	GC	GD	GE	GF	GG	GH	GI	GJ	GK	GL	GM	GN	GO	GP	GQ	GR	GS	GT	GU	GV	GW	GX	GY	GZ	HA	HB	HC	HD	HE	HF	HG	HH	HI	HJ	HK	HL	HM	HN	HO	HP	HQ	HR	HS	HT	HU	HV	HW	HX	HY	HZ	IA	IB	IC	ID	IE	IF	IG	IH	II	IJ	IK	IL	IM	IN	IO	IP	IQ	IR	IS	IT	IU	IV	IW	IX	IY	IZ	JA	JB	JC	JD	JE	JF	JG	JH	JI	IJ	JK	JL	JM	JN	JO	JP	JQ	JR	JS	JT	JU	JV	JW	JX	JY	JZ	KA	KB	KC	KD	KE	KF	KG	KH	KI	KJ	KK	KL	KM	KN	KO	KP	KQ	KR	KS	KT	KU	KV	KW	KX	KY	KZ	LA	LB	LC	LD	LE	LF	LG	LH	LI	LJ	LK	LL	LM	LN	LO	LP	LQ	LR	LS	LT	LU	LV	LW	LX	LY	LZ	MA	MB	MC	MD	ME	MF	MG	MH	MI	MJ	MK	ML	MM	MN	MO	MP	MQ	MR	MS	MT	MU	MV	MW	MX	MY	MZ	NA	NB	NC	ND	NE	NF	NG	NH	NI	NJ	NK	NL	NM	NN	NO	NP	NQ	NR	NS	NT	NU	NV	NW	NX	NY	NZ	OA	OB	OC	OD	OE	OF	OG	OH	OI	OJ	OK	OL	OM	ON	OO	OP	OQ	OR	OS	OT	OU	OV	OW	OX	OY	OZ	PA	PB	PC	PD	PE	PF	PG	PH	PI	PJ	PK	PL	PM	PN	PO	PP	PQ	PR	PS	PT	PU	PV	PW	PX	PY	PZ	QA	QB	QC	QD	QE	QF	QG	QH	QI	QJ	QK	QL	QM	QN	QO	QP	QQ	QR	QS	QT	QU	QV	QW	QX	QY	QZ	RA	RB	RC	RD	RE	RF	RG	RH	RI	RJ	RK	RL	RM	RN	RO	RP	RQ	RR	RS	RT	RU	RV	RW	RX	RY	RZ	SA	SB	SC	SD	SE	SF	SG	SH	SI	SJ	SK	SL	SM	SN	SO	SP	SQ	SR	SS	ST	SU	SV	SW	SX	SY	SZ	TA	TB	TC	TD	TE	TF	TG	TH	TI	TJ	TK	TL	TM	TN	TO	TP	TQ	TR	TS	TU	TV	TW	TX	TY	TZ	UA	UB	UC	UD	UE	UF	UG	UH	UI	UJ	UK	UL	UM	UN	UO	UP	UQ	UR	US	UT	UU	UV	UW	UX	UY	UZ	VA	VB	VC	VD	VE	VF	VG	VH	VI	VJ	VK	VL	VM	VN	VO	VP	VQ	VR	VS	VT	VU	VV	VW	VX	VY	VZ	WA	WB	WC	WD	WE	WF	WG	WH	WI	WJ	WK	WL	WM	WN	WO	WP	WQ	WR	WS	WT	WU	WV	WW	WX	WY	WZ	XA	XB	XC	XD	XE	XF	XG	XH	XI	XJ	XK	XL	XM	XN	XO	XP	XQ	XR	XS	XT	XU	XV	XW	XX	XY	XZ	YA	YB	YC	YD	YE	YF	YG	YH	YI	YJ	YK	YL	YM	YN	YO	YP	YQ	YR	YS	YT	YU	YV	YW	YX	YY	YZ	ZA	ZB	ZC	ZD	ZE	ZF	ZG	ZH	ZI	ZJ	ZK	ZL	ZM	ZN	ZO	ZP	ZQ	ZR	ZS	ZT	ZU	ZV	ZW	ZX	ZY	ZZ
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... dendritic



[illegible]

DOCUMENT NUMBER: 100-44470

TITLE: Prolongation of cardiac allograft survival using dendritic cells treated with NF- $\kappa$ B decoy

AUTHORS: Hannonakis, Nick; Bonham, C. Andrew; Qian, Shiguang;  
Zhou, Zhongyou; Peng, Lansha; Barnaba, Jo; Li, Wei;  
Inchomson, Angus W.; Fung, John J.; Robbins, Paul D.;  
Lu, Lina

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SOURCE: Molecular Therapy (2000), 10, Pt. 2, 430-437

CODEN: MTEORC; 1989; 1(2): 112-116

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DOCUMENT TYPE: ☐ Other, please specify: \_\_\_\_\_

LANGUAGE : DATE : \_\_\_\_\_

A13 Dendritic cells

There is a 1:1 relationship between any one of these vols. is assoc.

NOTE: NF- $\kappa$ B- $\alpha$  is a potent transcription factor in the

in its relation to the pathology has been assoc. with impaired NF

- .kappa.B- dependent transcription of costimulatory

genes as well as NF- $\kappa$ B translocation.

in the nucleus. In this report, we demonstrate that double-stranded

oligodeoxynucleotides cont. 1. binding sites for

NF- . kappa . B    NF- . kappa .

B QDN) are efficiently incorporated by the narrow-band-derived  $2^k$  and

specifically inhibit NF- $\kappa$ B-dependent

transcription of a reporter gene. Moreover, expression of the

oligonucleotide decays inhibited linear expansion of ISS - 100% inhibition

[illegible]

07 presenters with NF- $\kappa$ B

NF- $\kappa$ BNF- $\kappa$ B

$\frac{1}{2} \left( \frac{1}{2} \right) = \frac{1}{4}$

NF- $\kappa$ B

1. *Chlorophyll a* (Chl *a*)

Figure 1. The effect of the concentration of the *Agaricus bisporus* spores on the growth of *Agaricus bisporus* and *Agaricus bisporus* spores. The concentration of the spores was 10<sup>6</sup> spores/ml (A), 10<sup>7</sup> spores/ml (B), 10<sup>8</sup> spores/ml (C), and 10<sup>9</sup> spores/ml (D). The growth of *Agaricus bisporus* was measured by the diameter of the colony (mm) and the growth of *Agaricus bisporus* spores was measured by the diameter of the colony (mm).

[illegible][illegible]



LANGUAGE: English

AB

Molecular embryonic growth factor VEGF, produced by almost all tumor cells, affects the ability of bone marrow precursor cells (BPCs) to differentiate into functional dendritic cells (DC) during the early stages of their maturation. In this study we demonstrate specific binding of VEGF to BPC. This binding was efficiently competed by placenta growth factor (PIGF), a ligand reportedly specific to the Flt-1 receptor. The number of binding sites for VEGF increased during DC maturation in vitro as well with increased levels of mRNA for Flt-1. VEGF significantly inhibited release of tumor-infiltrating NF- $\kappa$ B-expressing DCs from tumor-bearing mice. This effect was blocked by transcription of the NF- $\kappa$ B inhibitor, I $\kappa$ B. In presence of VEGF, significantly reduced the number of DCs binding to NF- $\kappa$ B. NF- $\kappa$ B activation in BPCs was blocked with I $\kappa$ B-agonist. VEGF treatment significantly decreases in the mRNA for I $\kappa$ B-agonist, as well as for NF- $\kappa$ B. NF- $\kappa$ B binding to NF- $\kappa$ B inhibits in BPC at early stages of differentiation with an adenovirus expressing a dominant I $\kappa$ B-agonist inhibitor of NF- $\kappa$ B. Blockade of NF- $\kappa$ B repressed the pattern of effects obsd. with VEGF. Thus, NF- $\kappa$ B plays an important role in maturation of BPCs to DC, and VEGF activation of the Flt-1 receptor is able to block the activation of NF- $\kappa$ B in this system. Blockade of NF- $\kappa$ B activation in BPCs by tumor-derived factors may therefore be a mechanism by which tumor cells can directly down-modulate the ability of the immune system to generate effective antitumor immune responses.

L36 ANSWER 7 OF 8 "HELLO" CONFIDENTIAL 11-1-68

ACCESSION NUMBER: 19-0-071138 TAINUM  
DOCUMENT NUMBER: 12-0-07113  
TITLE: [REDACTED] ; STENOGRAPHIC  
[REDACTED] NF-kappa.

B. Gruppo "Laica": 1000  
Bianchi, Bruno; Bonaventura, Maria L.; Bianchi,  
Giovanni; Basso, Michele; Agresti, Emma; Fioretti,  
Maria L.; Fabbri, Italia

COMPOSITE SOURCE: Department of Experimental Medicine, University of  
Bologna, Bologna, 40138, Italy  
SOURCE: Immunity 1990, 9(3), 311-328  
ISSN: ISSN: 0950-2688  
EISSN: EISSN: 1474-7613

PUBLISHED: 1991-1994  
DOCUMENT TYPE: Technical  
LANGUAGE: English

AB The authors analyzed the expression of an IL-12 receptor by fresh dendritic cells (DC) and a DC line. Using RT-PCR, RT-PCR protection, and electrophoretic mobility shift assay (EMSA), they found that DC possess an IL-12 receptor with a small but distinct structural difference from that of T cells. IL-12 stimulates the activation of the receptor through activation of the NF- $\kappa$ B and not JAK/STAT pathway. The large proportion of the IL-12 receptor subunit, CR1, is located in the cytoplasmic binding sites with a high affinity for IL-12. The authors conclude that the IL-12 receptor is a novel type of receptor that is distinct from the IL-12 receptor expressed in T cells.

Author: [illegible]  
Title: [illegible]  
Source: [illegible]  
Date: [illegible]

Publisher: [illegible]

Document Type: [illegible]

Language: [illegible]

**AB** Interleukin-1 is induced in response to infection with bacteria or parasites or to bacterial constituents such as lipopolysaccharides (LPS) in monocytes/macrophages and **dendritic cells**, and also generated by the interaction between activated T cells and antigen-presenting cells via CD40-CD40 ligand (CD40L). Transcriptional analyses of p40 were carried out only using bacterial constituents such as LPS as stimuli. The transcriptional induction of p40 by CD40 ligation was characterized in a human B lymphoblastoid cell line, Daudi, and a human acute monocytic leukemia cell line, THP-1. These cells, stimulated by an agonistic monoclonal antibody against CD40, or by transfection with a CD40 expression vector, secreted p40 and also released p40 mRNA-expressing. Sequence analysis of the p40 promoter region identified a potential nuclear factor **NF- $\kappa$ B** binding sites conceptually between a murine and human. Electrophoretic mobility shift assay revealed that the potential **NF- $\kappa$ B** binding sequence which is located around 120 bp upstream of the transcription initiation site in murine and human p40 genes formed an **NF- $\kappa$ B** complex with nuclear extract from Daudi cells stimulated by CD40 ligation. Moreover, transfection of Daudi cells with the polymerase **NF- $\kappa$ B** binding sequence fused to a luciferase kinase/chloramphenicol acetyltransferase (CAT) reporter plasmid greatly induced CAT activity, but transfection with the poly(d, mutated **NF- $\kappa$ B** binding sequence did not. These results suggest that the **NF- $\kappa$ B** binding site located around 120 bp upstream of the transcription initiation site in murine and human p40 promoter regions could be important for the p40 induction by CD40 ligation via activation of **NF- $\kappa$ B**.

(FILE 'B44B' ENTERED AT 11:4:11 ON 01 NOV 2002)

FILE 'BIOSIS, MEDLINE, CABIUS, EMPAGE' ENTERED AT 11:4:11 ON 01 NOV 2002

L1 25547 NUCLEAR FACTOR KAPPA B  
L2 4421 TOLEROGEN?  
L3 44966 DENDRITIC CELL  
L4 15153 RIBOVYEN?  
L5 13 L1 AND L2  
L6 4 DUP REM L1 4 TITL WITH PRINCE  
L7 1 L1  
L8 4 DUP REM L1 4 TITL WITH PRINCE  
L9 2 L1 AND L2  
L10 1 L1  
L11 1 L1

FILE 'REAGENTS' ENTERED AT 1:16:11 ON 01 NOV 2002

L13 1 L1 2 L2 3 L3 4 L4 5 L5 6 L6 7 L7 8 L8 9 L9 10 L10 11 L11 12 L12 13 L13 14 L14 15 L15 16 L16 17 L17 18 L18 19 L19 20 L20 21 L21 22 L22 23 L23 24 L24 25 L25 26 L26 27 L27 28 L28 29 L29 30 L30 31 L31 32 L32 33 L33 34 L34 35 L35 36 L36 37 L37 38 L38 39 L39 40 L40 41 L41 42 L42 43 L43 44 L44 45 L45 46 L46 47 L47 48 L48 49 L49 50 L50 51 L51 52 L52 53 L53 54 L54 55 L55 56 L56 57 L57 58 L58 59 L59 60 L60 61 L61 62 L62 63 L63 64 L64 65 L65 66 L66 67 L67 68 L68 69 L69 70 L70 71 L71 72 L72 73 L73 74 L74 75 L75 76 L76 77 L77 78 L78 79 L79 80 L80 81 L81 82 L82 83 L83 84 L84 85 L85 86 L86 87 L87 88 L88 89 L89 90 L90 91 L91 92 L92 93 L93 94 L94 95 L95 96 L96 97 L97 98 L98 99 L99 100 L100 101 L101 102 L102 103 L103 104 L104 105 L105 106 L106 107 L107 108 L108 109 L109 110 L110 111 L111 112 L112 113 L113 114 L114 115 L115 116 L116 117 L117 118 L118 119 L119 120 L120 121 L121 122 L122 123 L123 124 L124 125 L125 126 L126 127 L127 128 L128 129 L129 130 L130 131 L131 132 L132 133 L133 134 L134 135 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FILE 'BIOSIS, MEDLINE, CASIMP, EMBASE' ENTERED AT 11:34:46 ON 21 NOV 2002

L1 1 14 17 TRAP PAIR B KALPA P  
L2 41402 NE KALPA P  
L3 4401 TRIPB BUN  
L4 18183 KIPOTVW  
L5 183175 OLIGONUCLEOTIDE  
L6 77082 ANTISENS?  
L7 622 L1 AND L6  
L8 1 L7 AND L3  
L9 1054 L2 AND L6  
L10 3 L9 AND L3  
L11 1 DUP REM L1 (6 UTILITIES REMOVED)  
L12 18 L1 AND L4 AND L3  
L13 1 L1 AND L2  
L14 1 L1 AND L3 AND L2  
L15 1 L1 AND L2  
L16 80 L1 AND L2  
L17 44900 LPRINTING TEST  
L18 1 L1 AND L1 AND L1  
L19 1 DUP REM L1 (1 DUPLICATE REMOVED)  
L20 1 L1 AND L1 AND L17  
L21 1 L1 REM L1 (6 UTILITIES REMOVED)

FILE 'BIOSIS, MEDLINE, CASIMP, EMBASE' ENTERED AT 11:48:58 ON 21 NOV 2002

L2 7 L1 AND L3 AND L1  
L3 5 DUP REM L2 (2 DUPLICATES REMOVED)  
L4 10 L17 AND L3 AND L2  
L5 6 DUP REM L24 (4 DUPLICATES REMOVED)  
L6 0 L16 AND L3 AND L1  
L7 0 L16 AND L3 AND L2







REFERENCE COUNT: 2. THERE ARE 2. CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PATENT INFORMATION:

L.; Kalande, P. H. K.; Williams, I. M. Roy; Wood, William L.; Yarbrough, Larry, J.

FOOT INT. April, 1968 pp.  
COLON: LINE 1.

Fig. 1.  $\alpha$  and  $\beta$  components.

F.M.D. 155

1

[illegible]



AU 2001071973	AC 2002090681
US 2002090681	AC 2002090681

PRIORITY AREA. INFO.:

[illegible]



AB Antisense oligonucleotides, 100011, 100012 are provided  
 100013 : 100014 for the synthesis of a BAMP-oligonucleotide  
 100015 nuclear factor-kappa B, 100016  
 100017 known to induce T-47D growth and dendritic cell  
 100018 induction. The oligos, oligonucleotides 100019, particularly antisense  
 100020 oligonucleotides, 100021 100022 100023 100024 100025 100026 100027 100028 100029 100030 100031 100032 100033 100034 100035 100036 100037 100038 100039 100040 100041 100042 100043 100044 100045 100046 100047 100048 100049 100050 100051 100052 100053 100054 100055 100056 100057 100058 100059 100060 100061 100062 100063 100064 100065 100066 100067 100068 100069 100070 100071 100072 100073 100074 100075 100076 100077 100078 100079 100080 100081 100082 100083 100084 100085 100086 100087 100088 100089 100090 100091 100092 100093 100094 100095 100096 100097 100098 100099 100100 100101 100102 100103 100104 100105 100106 100107 100108 100109 100110 100111 100112 100113 100114 100115 100116 100117 100118 100119 100120 100121 100122 100123 100124 100125 100126 100127 100128 100129 100130 100131 100132 100133 100134 100135 100136 100137 100138 100139 100140 100141 100142 100143 100144 100145 100146 100147 100148 100149 100150 100151 100152 100153 100154 100155 100156 100157 100158 100159 100160 100161 100162 100163 100164 100165 100166 100167 100168 100169 100170 100171 100172 100173 100174 100175 100176 100177 100178 100179 100180 100181 100182 100183 100184 100185 100186 100187 100188 100189 100190 100191 100192 100193 100194 100195 100196 100197 100198 100199 100200 100201 100202 100203 100204 100205 100206 100207 100208 100209 100210 100211 100212 100213 100214 100215 100216 100217 100218 100219 100220 100221 100222 100223 100224 100225 100226 100227 100228 100229 100230 100231 100232 100233 100234 100235 100236 100237 100238 100239 100240 100241 100242 100243 100244 100245 100246 100247 100248 100249 100250 100251 100252 100253 100254 100255 100256 100257 100258 100259 100260 100261 100262 100263 100264 100265 100266 100267 100268 100269 100270 100271 100272 100273 100274 100275 100276 100277 100278 100279 100280 100281 100282 100283 100284 100285 100286 100287 100288 100289 100290 100291 100292 100293 100294 100295 100296 100297 100298 100299 100300 100301 100302 100303 100304 100305 100306 100307 100308 100309 100310 100311 100312 100313 100314 100315 100316 100317 100318 100319 100320 100321 100322 100323 100324 100325 100326 100327 100328 100329 100330 100331 100332 100333 100334 100335 100336 100337 100338 100339 100340 100341 100342 100343 100344 100345 100346 100347 100348 100349 100350 100351 100352 100353 100354 100355 100356 100357 100358 100359 100360 100361 100362 100363 100364 100365 100366 100367 100368 100369 100370 100371 100372 100373 100374 100375 100376 100377 100378 100379 100380 100381 100382 100383 100384 100385 100386 100387 100388 100389 100390 100391 100392 100393 100394 100395 100396 100397 100398 100399 100400 100401 100402 100403 100404 100405 100406 100407 100408 100409 100410 100411 100412 100413 100414 100415 100416 100417 100418 100419 100420 100421 100422 100423 100424 100425 100426 100427 100428 100429 100430 100431 100432 100433 100434 100435 100436 100437 100438 100439 100440 100441 100442 100443 100444 100445 100446 100447 100448 100449 100450 100451 100452 100453 100454 100455 100456 100457 100458 100459 100460 100461 100462 100463 100464 100465 100466 100467 100468 100469 100470 100471 100472 100473 100474 100475 100476 100477 100478 100479 100480 100481 100482 100483 100484 100485 100486 100487 100488 100489 100490 100491 100492 100493 100494 100495 100496 100497 100498 100499 100500 100501 100502 100503 100504 100505 100506 100507 100508 100509 100510 100511 100512 100513 100514 100515 100516 100517 100518 100519 100520 100521 100522 100523 100524 100525 100526 100527 100528 100529 100530 100531 100532 100533 100534 100535 100536 100537 100538 100539 100540 100541 100542 100543 100544 100545 100546 100547 100548 100549 100550 100551 100552 100553 100554 100555 100556 100557 100558 100559 100560 100561 100562 100563 100564 100565 100566 100567 100568 100569 100570 100571 100572 100573 100574 100575 100576 100577 100578 100579 100580 100581 100582 100

REFERENCE FORM: 1. THERE ARE NO OTHER REFERENCES AVAILABLE FOR THIS  
REF ID: A66-11414-1. NO OTHER REFERENCES AVAILABLE IN THE RE FORM.

L19 ANSWER # OF 11 CASING COPYRIGHT 1991 ACS  
 ACCESSION NUMBER: 101:011744 CASREF  
 DOCUMENT NUMBER: 101:0607  
 TITLE: Expression of different NF-kappa.B pathway genes in  
 dendritic cells (DCs) or macrophages  
 assessed by gene expression profiling  
 AUTHCR(S): Baltathakis, Ioannis; Alcantara, Orlando; Baldt, David  
 H.  
 CORPORATE SOURCE: Medicine/Hematology, University of Texas Health  
 Science Center, San Antonio, TX, 78229-3900, USA  
 SOURCE: Journal of Cellular Biochemistry 2001, 83:26,  
 451-457  
 WHEN: 2001; WHEN: 2001-01-01  
 PUBLISHER: Wiley-Liss, Inc.  
 1 COMMENT: 1  
 101:0607

AB NF- $\kappa$ B plays a central role in the regulation of gene expression in the differentiation of T lymphocytes into **dendritic cells** (DCs). In this process, the transcription factor NF- $\kappa$ B is activated in dendritic precursor cells. Recent studies of the expression pattern of NF- $\kappa$ B pathway and their inhibitors (I $\kappa$ B $\alpha$ , Bcl-2) suggest that these molecules regulate this differentiation process in a complex manner. To investigate differential gene expression between naive splenic T cells and DCs, we used a 12,000 cDNA available gene microarrays (cDNA Array Kit), which included four of the NF- $\kappa$ B/Rel family genes (p50/p105, p52/p102, Bcl-2, and I $\kappa$ B $\alpha$ ) and several genes either in the NF- $\kappa$ B signal transduction pathway or under transcriptional control of NF- $\kappa$ B/Rel factors. The dendritic precursor and DCs, as well as adherent peripheral blood monocytes were cultured with IL-1 $\alpha$ , IL-1 $\beta$ , IL-4 and IL-6 for 4 days. DCs and adherent peripheral monocytes were treated with 10 ng/ml lipopolysaccharide (LPS) for the last 24 h of culture. In naive splenic T cells, we observed a 10-fold increase in p50, p52 and radiolabeled with oligonucleotides (p50-3'UTR, p52-3'UTR, Bcl-2-3'UTR, I $\kappa$ B $\alpha$ -3'UTR) and a 2-fold increase in Bcl-2-3'UTR. In DCs, we observed a 10-fold increase in p50-3'UTR, a 5-fold increase in p52-3'UTR, a 10-fold increase in Bcl-2-3'UTR, and a 10-fold increase in I $\kappa$ B $\alpha$ -3'UTR. In adherent peripheral monocytes, we observed a 10-fold increase in p50-3'UTR, a 5-fold increase in p52-3'UTR, a 10-fold increase in Bcl-2-3'UTR, and a 10-fold increase in I $\kappa$ B $\alpha$ -3'UTR. These results suggest that NF- $\kappa$ B plays a central role in the differentiation of T lymphocytes into DCs.

significantly upregulated in mature ICs compared to macrophages. The strongest difference was seen for c-rel, NF- $\kappa$ B doctns. of c-rel, RelB, and I $\kappa$ B mRNAs confirmed these observations. Among the 32 NF- $\kappa$ B/ $\kappa$ B pathway genes, 14 were upregulated in mature ICs compared to macrophages. These genes were I $\kappa$ B.alpha., I $\kappa$ B.beta., NIK, IKAM-1, E-selectin, E-selectin, TNF.alpha., TNFR2, TNFAIP3, IL-1.alpha., IL-1 $\beta$ , IL-1 $\kappa$ , IRAK, and TANK. By contrast, only myd-1 (myeloid differentiation protein 1) was upregulated in macrophages compared to ICs. NF- $\kappa$ B pathway genes upregulated in ICs compared to macrophages were constitutively expressed in monocytes that selectively downregulated during maturation into IC differentiation. IL-1 and IL-1 $\beta$  were expressed in a subset of these genes in macrophages and did not change with maturation of ICs in mature macrophages. The upregulated genes in macrophages compared to ICs, such as c-rel, are selectively expressed during differentiation of monocytes towards DCs. Moreover, only a subset of genes were upregulated both with activation of different NF- $\kappa$ B signaling pathways in DCs and macrophages and with expression of a subset of genes in ICs that are transcriptionally regulated by NF- $\kappa$ B/ $\kappa$ B factors. The results indicate the ability of the NF- $\kappa$ B pathway to respond to differentiation stimuli by activating in a cell-specific manner unique signaling pathways and subsets of NF- $\kappa$ B target genes.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:904923 CAPLUS

DOCUMENT NUMBER: 134:161862

TITLE: A Toll-like receptor recognizes bacterial DNA

AUTHOR(S): Hemmi, Hiroaki; Takahashi, Kazuo; Kawai, Taro; Kishimoto, Tsuneyasu; Saito, Shintaro; Sato, Hiroaki; Nakajima, Makoto; Hoshino, Katsuki; Tanabe, Tetsuo; Takeda, Kiyoshi; Akira, Shizuo

CORPORATE SOURCE: Department of Pathology, Osaka University, 1-8 Yamadaoka, Suita, Osaka, 565-0871, Japan; Osaka University and the Research Center for Immunology and Technology, Suita, Osaka, 565-0871, Japan

SOURCE: Nature, London, England, 406(6813), 740-745

ISSN: 0028-0836; ISSN: 0028-0836

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB DNA is an important stimulatory effector in mammalian immune cells, which depends on the presence of unmethylated CpG dinucleotides in the bacterial DNA. In contrast, mammalian DNA has a low frequency of CpG dinucleotides, and these are mostly methylated; therefore, mammalian DNA does not have immune-stimulatory activity. CpG DNA induces a strong T-helper-1-like inflammatory response. Accumulating evidence has revealed the therapeutic potential of CpG DNA as adjuvants for vaccination strategies for cancer, allergy and infectious diseases. Despite its promising clinical use, the mol. mechanism by which CpG DNA activates immune cells remains unclear. Here the authors show that cellular responses to CpG DNA are mediated by a Toll-like receptor, TLR4. TLR4-deficient mice do not show any response to CpG DNA, and adoptive transfer of TLR4-deficient dendritic cells into wild-type mice abrogates the response to CpG DNA. The induction of TLR4-deficient dendritic cells by CpG DNA was also blocked in TLR4-deficient mice, suggesting that TLR4 is a key receptor for CpG DNA. These results suggest that TLR4 is a key receptor for CpG DNA from bacteria.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2002 ACS



ACCESSION NUMBER: 00000000000000000000  
JOURNAL NUMBER: 00000000000000000000  
TITLE: Dendritic cells in the development of allergic diseases  
AUTHOR: Li, Wei; Wang, J.; Zhang, L.; Zhang, Y.; Zhang, X.; Zhang, Z.; Zhang, H.; Zhang, J.; Zhang, K.; Zhang, L.; Zhang, M.; Zhang, N.; Zhang, P.; Zhang, Q.; Zhang, R.; Zhang, S.; Zhang, T.; Zhang, U.; Zhang, V.; Zhang, W.; Zhang, X.; Zhang, Y.; Zhang, Z.; Zhang, A.; Zhang, B.; Zhang, C.; Zhang, D.; Zhang, E.; Zhang, F.; Zhang, G.; Zhang, H.; Zhang, I.; Zhang, J.; Zhang, K.; Zhang, L.; Zhang, M.; Zhang, N.; Zhang, O.; Zhang, P.; Zhang, Q;  
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry,  
University of Pittsburgh, Pittsburgh, PA, 15261, USA  
SOURCE: Molecular Therapy 19(2007), Vol. Pt. 1, 430-437  
PUBIDEN: PUBMED; ISSN: 1547-0016  
PUBLISHER: Academic Press  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Dendritic cells (DC) classically promote immune responses but can be manipulated to induce antigen-specific hyporesponsiveness in vitro. The expression of costimulatory moles, CD80, CD86, CD80) at the DC cell surface correlates with their ability to induce or suppress immune responses. Expression of these moles is associated with NF- $\kappa$ B-dependent transcription of their genes. DC transcription has been associated with induced NF- $\kappa$ B-dependent transcription of a variety of genes as well as NF- $\kappa$ B translocation to the nucleus. In this report, we demonstrate that double-stranded oligodeoxynucleotides (ODN) containing motifs for NF- $\kappa$ B (NF- $\kappa$ B ODN) are effectively up-regulated by bone marrow-derived DC and up-regulate NF- $\kappa$ B-dependent transcription of a reporter gene. Moreover, exposure of DC to the oligonucleotide decoys inhibits oligodeoxynucleotide (ODN)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF- $\kappa$ B ODN selectively suppressed the cell-surface expression of costimulatory moles, without interfering with MHC class I or class II expression. Furthermore, NF- $\kappa$ B ODN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was associated with inhibition of Th1-type cytokine prodn. Finally, infusion of NF- $\kappa$ B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immune suppression. Specific interference with NF- $\kappa$ B and other transcriptional pathways involved in immune stimulation in DC using ODN may provide a therapeutic means to promote tolerance induction in heart transplantation. (Supported by the National Institutes of Health, Bethesda, MD, and the American Heart Association, Dallas, TX).

the 1990s, the number of people in the world who are illiterate has increased from 1.2 billion to 1.5 billion. The number of illiterate people in the world is expected to increase to 1.7 billion by the year 2015. The number of illiterate people in the world is expected to increase to 1.7 billion by the year 2015.

TITLE: *Effect of 3'-terminal polyphosphate-containing oligonucleotides on the binding of nuclear factor .kappa.B*

AUTHOR: T.H.; Koppelman, J.; Rutter, T.L.; Bellinger C.C.; Rudy  
J.R.; Hoot T.L.; Conrad E.J.

CORPORATE OFFICE: Mr. R. L. Jorgensen, Vice-President of Viradil, HOT Box 400000,  
Charles C. Smith Co. Building, 1001, East Tennessee, VA  
11/10/71, 11/10/71

SOURCE: NEW YORK, RECEIVED, APRIL 1967. - NY 100-81110

[illegible]

COUNTRY: THAILAND

DOCUMENIT DATED: 07/06/2008

2000, 2001, 2002, 2003, 2004, 2005, 2006, 2007, 2008, 2009, 2010, 2011, 2012, 2013, 2014, 2015, 2016, 2017, 2018, 2019, 2020, 2021, 2022, 2023, 2024, 2025, 2026, 2027, 2028, 2029, 2030, 2031, 2032, 2033, 2034, 2035, 2036, 2037, 2038, 2039, 2040, 2041, 2042, 2043, 2044, 2045, 2046, 2047, 2048, 2049, 2050, 2051, 2052, 2053, 2054, 2055, 2056, 2057, 2058, 2059, 2060, 2061, 2062, 2063, 2064, 2065, 2066, 2067, 2068, 2069, 2070, 2071, 2072, 2073, 2074, 2075, 2076, 2077, 2078, 2079, 2080, 2081, 2082, 2083, 2084, 2085, 2086, 2087, 2088, 2089, 2090, 2091, 2092, 2093, 2094, 2095, 2096, 2097, 2098, 2099, 2100, 2101, 2102, 2103, 2104, 2105, 2106, 2107, 2108, 2109, 2110, 2111, 2112, 2113, 2114, 2115, 2116, 2117, 2118, 2119, 2120, 2121, 2122, 2123, 2124, 2125, 2126, 2127, 2128, 2129, 2130, 2131, 2132, 2133, 2134, 2135, 2136, 2137, 2138, 2139, 2140, 2141, 2142, 2143, 2144, 2145, 2146, 2147, 2148, 2149, 2150, 2151, 2152, 2153, 2154, 2155, 2156, 2157, 2158, 2159, 2160, 2161, 2162, 2163, 2164, 2165, 2166, 2167, 2168, 2169, 2170, 2171, 2172, 2173, 2174, 2175, 2176, 2177, 2178, 2179, 2180, 2181, 2182, 2183, 2184, 2185, 2186, 2187, 2188, 2189, 2190, 2191, 2192, 2193, 2194, 2195, 2196, 2197, 2198, 2199, 2200, 2201, 2202, 2203, 2204, 2205, 2206, 2207, 2208, 2209, 2210, 2211, 2212, 2213, 2214, 2215, 2216, 2217, 2218, 2219, 2220, 2221, 2222, 2223, 2224, 2225, 2226, 2227, 2228, 2229, 2230, 2231, 2232, 2233, 2234, 2235, 2236, 2237, 2238, 2239, 2240, 2241, 2242, 2243, 2244, 2245, 2246, 2247, 2248, 2249, 2250, 2251, 2252, 2253, 2254, 2255, 2256, 2257, 2258, 2259, 2260, 2261, 2262, 2263, 2264, 2265, 2266, 2267, 2268, 2269, 2270, 2271, 2272, 2273, 2274, 2275, 2276, 2277, 2278, 2279, 2280, 2281, 2282, 2283, 2284, 2285, 2286, 2287, 2288, 2289, 2290, 2291, 2292, 2293, 2294, 2295, 2296, 2297, 2298, 2299, 2300, 2301, 2302, 2303, 2304, 2305, 2306, 2307, 2308, 2309, 2310, 2311, 2312, 2313, 2314, 2315, 2316, 2317, 2318, 2319, 2320, 2321, 2322, 2323, 2324, 2325, 2326, 2327, 2328, 2329, 2330, 2331, 2332, 2333, 2334, 2335, 2336, 2337, 2338, 2339, 2340, 2341, 2342, 2343, 2344, 2345, 2346, 2347, 2348, 2349, 2350, 2351, 2352, 2353, 2354, 2355, 2356, 2357, 2358, 2359, 2360, 2361, 2362, 2363, 2364, 2365, 2366, 2367, 2368, 2369, 2370, 2371, 2372, 2373, 2374, 2375, 2376, 2377, 2378, 2379, 2380, 2381, 2382, 2383, 2384, 2385, 2386, 2387, 2388, 2389, 2390, 2391, 2392, 2393, 2394, 2395, 2396, 2397, 2398, 2399, 2400, 2401, 2402, 2403, 2404, 2405, 2406, 2407, 2408, 2409, 2410, 2411, 2412, 2413, 2414, 2415, 2416, 2417, 2418, 2419, 2420, 2421, 2422, 2423, 2424, 2425, 2426, 2427, 2428, 2429, 2430, 2431, 2432, 2433, 2434, 2435, 2436, 2437, 2438, 2439, 2440, 2441, 2442, 2443, 2444, 2445, 2446, 2447, 2448, 2449, 2450, 2451, 2452, 2453, 2454, 2455, 2456, 2457, 2458, 2459, 2460, 2461, 2462, 2463, 2464, 2465, 2466, 2467, 2468, 2469, 2470, 2471, 2472, 2473, 2474, 2475, 2476, 2477, 2478, 2479, 2480, 2481, 2482, 2483, 2484, 2485, 2486, 2487, 2488, 2489, 2490, 2491, 2492, 2493, 2494, 2495, 2496, 2497, 2498, 2499, 2500, 2501, 2502, 2503, 2504, 2505, 2506, 2507, 2508, 2509, 2510, 2511, 2512, 2513, 2514, 2515, 2516, 2517, 2518, 2519, 2520, 2521, 2522, 2523, 2524, 2525, 2526, 2527, 2528, 2529, 2530, 2531, 2532, 2533, 2534, 2535, 2536, 2537, 2538, 2539, 2540, 2541, 2542, 2543, 2544, 2545, 2546, 2547, 2548, 2549, 2550, 2551, 2552, 2553, 2554, 2555, 2556, 2557, 2558, 2559, 2560, 2561, 2562, 2563, 2564, 2565, 2566, 2567, 2568, 2569, 2570, 2571, 2572, 2573, 2574, 2575, 2576, 2577, 2578, 2579, 2580, 2581, 2582, 2583, 2584, 2585, 2586, 2587, 2588, 2589, 2590, 2591, 2592, 2593, 2594, 2595, 2596, 2597, 2598, 2599, 2600, 2601, 2602, 2603, 2604, 2605, 2606, 2607, 2608, 2609, 2610, 2611, 2612, 2613, 2614, 2615, 2616, 2617, 2618, 2619, 2620, 2621, 2622, 2623, 2624, 2625, 2626, 2627, 2628, 2629, 2630, 2631, 2632, 2633, 2634, 2635, 2636, 2637, 2638, 2639, 2640, 2641, 2642, 2643, 2644, 2645, 2646, 2647, 2648, 2649, 2650, 2651, 2652, 2653, 2654, 2655, 2656, 2657, 2658, 2659, 2660, 2661, 2662, 2663, 2664, 2665, 2666, 2667, 2668, 2669, 2670, 2671, 2672, 2673, 2674, 2675, 2676, 2677, 2678, 2679, 2680, 2681, 26

# SUMMARY LANGUAGE: English

**AB** Background. DNA containing the CpG motif is associated with immunomodulation of the innate immune response. Pre-exposure of macrophages to CpG DNA elicits a hyporesponsiveness to subsequent lipopolysaccharide (LPS) stimulation. We tested the hypothesis that this effect is due to decreased nuclear translocation of **nuclear factor** **kappa.B** (NK-.kappa.B). Methods. Murine macrophage-like RAW 264.7 cells were incubated with 1.0  $\mu$ M CpG DNA-containing **oligonucleotides** (CpG ODN) for 1 h and then restimulated with 1.0  $\mu$ M LPS for 1 h. Cells were cotransfected with an NF-.kappa.B sensitive luciferase reporter construct and a pRL-TK-luciferase plasmid. Cytoplasmic and nuclear extracts were assayed for NF-.kappa.B and I.kappa.B kinase activity by  $\gamma$ -irradiation and Western blotting, and I.kappa.B kinase activity by Western blotting and kinase assay. Results. NF-.kappa.B kinase activity was increased as demonstrated by luciferase activity assay in the prolonged CpG ODN pretreatment groups. Unlike endotoxin tolerance, CpG ODN pre-exposure increased cytoplasmic phospho-I.kappa.B.  $\alpha$  and did not abrogate mitogen-activated protein kinase activity. Conclusions. In macrophages, exposure to CpG DNA increases expression of the inhibitory p50 NF-.kappa.B homodimer and decreases NF-.kappa.B activity without inhibition of I.kappa.B kinases. Mitogen-activated protein kinase activity remains intact. Understanding these interactions between different toll receptor ligands may provide insight into novel therapeutic modalities.



NF- $\kappa$ B signaling pathway has been implicated in the differentiation of myeloid cells either dendritic cells (DCs) or macrophages, as well as in the maturation of DCs from antigen-processing to antigen-presenting cells. Recent studies of the expression pattern of Rel proteins and their inhibitors (IkappaBs), suggest that their regulation during this differentiation process is transcriptional. To investigate differential gene expression between macrophages and DCs, we used commercially available low-risk arrays (GeneChip Kit), which included 60 members of NF- $\kappa$ B/Rel family genes (p105/p50, p50/p51, RelB, c-rel, bcl-2, and others). We observed that the NF- $\kappa$ B signal transduction pathway was up-regulated in mature DCs compared to macrophages. In contrast, macrophage-specific genes such as mannose-binding lectin, iNOS, and inducible peroxylase were down-regulated with NF- $\kappa$ B or TNF- $\alpha$ /IL-1 + IL-4 respectively treatment. Using RT-PCR and Northern experiments, macrophages were treated with LPS or interferon- $\gamma$  for the last 48 h of culture to induce activation. Cells were harvested after 7 days, mRNA was prepared and reverse-transcribed. Ikappa-B- $\alpha$ , B-actin, then hybridized to gene arrays containing specific gene probes, beta-actin and GAPDH or PUC18 oligonucleotides served as positive or negative controls, respectively. The expression of all four NF- $\kappa$ B/Rel family genes examined was significantly upregulated in maturing DCs compared to macrophages. The strongest difference was observed for c-rel. RT-PCR determinations of c-rel, RelB, and p105 mRNAs confirmed these observations. Among the 32 NF- $\kappa$ B/Rel pathway genes, 14 were upregulated in mature DCs compared to macrophages. These genes were IkappaB $\alpha$ , IKK-beta, NIK, ICAM-1, P-selectin, E-selectin, TNF-alpha, TNFR2, TNFAIP3, IL-1alpha, IL-1R1, IL-1R2, IRAK, and TANK. By contrast, only MCP-1 (monocyte chemotactic protein 1) was upregulated in macrophages compared to DCs. NF- $\kappa$ B pathway genes upregulated in DCs compared to macrophages were constitutively expressed in macrophages then selectively downregulated during macrophage but not DC differentiation. LPS did not increase expression of most of these genes in macrophages but LPS did induce significant upregulation of several genes. We found that NF-kappaB pathway genes, including c-rel, are selectively up-regulated through differentiating pathways. Therefore, this differential expression is associated with activation of different NF-kappaB signal transduction pathways in DCs and macrophages and with expression of unique subsets of genes in DCs that are transcriptionally targeted by NF-kappaB/Rel factors. The results illustrate the ability of the NF-kappaB pathway to respond to differentiation stimuli by activating in a cell-specific manner unique signaling pathways and subsets of NF-kappaB target genes.

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L21 ANSWER 3 OF 15 NEPLINE NEPLINE NEPLINE  
ACCESSION NUMBER: 2000419045 NEPLINE  
DOCUMENT NUMBER: 2000419045 PubMed ID: 1140464  
TITLE: Preliminary report on the effect of dendritic cells  
dendritic cells treated with CD-80 on the  
effect of dendritic cells on the  
COMMENT: Program ID: NEPLINE NEPLINE NEPLINE  
AUTHOR: NEPLINE NEPLINE NEPLINE  
JOURNAL: NEPLINE NEPLINE NEPLINE  
CONTRACT NUMBER: NEPLINE NEPLINE NEPLINE  
SOURCE: NEPLINE NEPLINE NEPLINE  
PUB. NUMBER: NEPLINE NEPLINE NEPLINE  
PUB. DATE: NEPLINE NEPLINE NEPLINE

Dendritic cells (DC) are the most potent antigen-presenting cells (APC) in the immune system. These cells express a variety of costimulatory molecules (CD40, CD80, CD86) on their surface that correlates with their capacity to induce or suppress immune responses. Expression of these molecules is associated with NF- $\kappa$ B-dependent transcription of their genes. DC tolerogenicity has been associated with impaired NF- $\kappa$ B-dependent transcription of costimulatory genes as well as NF- $\kappa$ B translocation to the nucleus. In this report, we demonstrate that double-stranded oligodeoxyribonucleotides containing binding sites for NF- $\kappa$ B (NF- $\kappa$ B ODN) are efficiently incorporated by bone marrow-derived DC and specifically inhibit NF- $\kappa$ B-dependent transcription of a reporter gene. Moreover, exposure of DC to the oligonucleotide decoys inhibited lipopolysaccharide (LPS)-induced nitric oxide production, a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF- $\kappa$ B ODN selectively suppressed the cell-surface expression of costimulatory molecules without interfering with MHC class I or class II expression. Furthermore, NF- $\kappa$ B ODN inhibited DC-induced anti-specific responses in vivo. This effect was cell-type specific, and this was associated with inhibition of all-type cytokine production. Finally, infusion of NF- $\kappa$ B ODN-treated DC prior to transplantation in allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of transplant rejection. Specific interference with NF- $\kappa$ B-dependent transcriptional pathways involved in immune stimulation in DC using ODN decoys might provide the means to promote tolerance induction in heart transplantation.

polyclonal anti-CD3, anti-CD4, anti-CD8 (T3AM-3) or anti-CD99 mAb. The MEM-49-mediated apoptosis of Tc-1 cells was also inhibited by the overexpression of a specific inhibitor, Baxx. T4i-mediated apoptosis was preceded by the repression of the DNA binding activity of the transcription factor NF- $\kappa$ B. RNA array screening revealed that the expression of several genes encoding apoptosis-regulating proteins, including 14-3-3 proteins and the granulocyte macrophage colony-stimulating factor (GM-CSF) receptor beta-subunit, was repressed in TF-1 cells bound to immobilized MEM-49. The down-regulation of 14-3-3 proteins and GM-CSF receptor beta was accompanied by translocation of the proapoptotic protein Bad to the mitochondria. These results suggest that engagement of CD43 may, presumably through the repressing transcription, initiate a Bad-dependent apoptotic pathway.

L21 ANSWER 5 OF 15  
ADDRESS: L21@...  
DOCUMENT NUMBER: ...  
TITLE: ...  
AUTHOR: ...  
CORPORATE SOURCE: ...  
CONTRACT NUMBER: ...  
SOURCE: ...  
PUB. COUNTRY: ...  
DOCUMENT TYPE: ...  
LANGUAGE: ...  
FILE SEGMENT: ...  
ENTRY MONTH: ...  
ENTRY DATE: ...

AB 1. In the present study, we have shown that the growth and differentiation of dendritic dendrites is under the control of factors that are regulated by innervation. In the present study, we have shown that dendrites of specialized cells such as **dendritic cells**, for which their receptors respond to fragments of extracellular molecules is not known. We found that the expression of **Toll-like receptor 4** in **dendritic cells** might be related to the ability of fragments of heparan sulfate polysaccharide. **Dendritic cells** were found to mature in response to heparan sulfate as revealed by a stimulatory protein expression, morphologic, and functional activation, but this maturation was absent when **Toll-like receptor 4** was silenced or inhibited. These findings suggest that **Toll-like receptors** in dendrites may monitor tissue well-being by recognizing fragments of extracellular molecules.

L21 ANSWER 6 OF 15 MEDLINE  
 ACCESSION NUMBER: 2002-03702 MEDLINE  
 DOCUMENT NUMBER: 100-000-100-100-100  
 TITLE: Morphology of dendritic cells :  
 a review of the literature  
 AUTHOR: [illegible]  
 CITATION: [illegible]  
 INITIALS: [illegible]  
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PUB. # 1187.  
 DOCUMENT TYPE: Review Article; MEDLINE ABSTRACT  
 \*\*Review\*\*  
 Language: English, Unpub.  
 LANGUAGE: English  
 FILE # 00000  
 ENTRY MONTH: January 1999  
 ENTRY YEAR: 1999  
 Entered EMTN: 21 01 99  
 Last Updated on EMTN: 21 01 99  
 Entered Medline: 20020213

**AB Dendritic cells** DC constitute a complex system of uniquely well-equipped antigen-presenting cells that initiate and regulate immune responses. Extensive recent studies have improved our understanding of DC development, differentiation, activation, and function. DC exist as distinct subsets that differ in their lineage, maturation, surface molecule expression, and biological function. These factors work to determine the T-cell polarizing signals and type of T-cell response—T helper 1, T helper 2, or T regulatory—induced by DC. Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T-regulatory cell activity, and promotion of activated T-cell apoptosis. Understanding of the biology of the molecular basis of DC biology, including their phenotype, maturation, emerging information suggests that dendritic cells, by virtue of their expression of death-inducing ligands, support a variety of cell death, microenvironmental factors (in particular, anti-apoptotic and immunosuppressive cytokines), and inhibition of dendritic maturation and secretory proteins (e.g., nuclear factor-kappaB) can impact tolerance potential. Manipulation of DC by control of their maturation and differentiation, or genetic engineering of these cells to express immunosuppressive molecules, offers potential for therapy of allograft rejection and autoimmune disease. In this brief overview, we outline principles and methods for generation of "tolerogenic" DC and outcomes that have been reported in experimental models. Space constraints limit literature citations.

L21 ANSWER 3 OF 15 MEDLINE  
 ACCESSION NUMBER: 200211821 MEDLINE  
 DOCUMENT NUMBER: 21900697 PubMed ID: 12447606  
 TITLE: DNA array and biological characterization of the impact of the natural killer cell on dendritic cells in the rejection of allogeneic heart transplantation.  
 AUTHOR: [illegible]  
 CORPORA # 1187: [illegible]  
 SOURCE: [illegible]  
 PUB. # 1187.  
 DOCUMENT TYPE: Review Article; MEDLINE ABSTRACT  
 LANGUAGE: English  
 FILE # 00000  
 ENTRY MONTH: January 1999  
 ENTRY YEAR: 1999  
 ENTRY DATE: Entered EMTN: 21 01 99  
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 Entered Medline: 20020213

**AB** We systematically investigated the impact of the natural killer cell levels of **dendritic cells** on the rejection of allogeneic heart transplantation. We systematically investigated the impact of the natural killer cell levels of dendritic cells on the rejection of allogeneic heart transplantation. We systematically investigated the impact of the natural killer cell levels of dendritic cells on the rejection of allogeneic heart transplantation.

[illegible]121 ANSWER: 100. The number of possible outcomes is  $2^6 = 64$ . The number of outcomes in which the sum of the numbers is 100 is 1. The probability is  $\frac{1}{64}$ .

ACCESSION NUMBER: 2012:049719 CREF:18

DOCUMENT NUMBER: 13-193759

TITLE: Potential role of phosphatidylinositol 3 kinase, rather than DNA-dependent protein kinase, in CpG DNA-induced immune activation

AUTHOR(S): Ishii, Ken J.; Takeshita, Fumihiko; Gursel, Isan;  
Gursel, Mayda; Chover, Jacqueline; Wassenaar, Andre;  
Klinman, Dennis M.

CORPORATE SOURCE: Section of Retroviral Immunology, Division of Viral Products, Center for Biologics Evaluation and Research, Food and Drug Administration, Bethesda, Institutes of Health, Bethesda, MD, 20892, USA

SOURCE: *U.S. Census Bureau, "Population in the United States, 1900-1990," 1991, 1992, 1993, 1994.*

FOOTNOTES: 11-14: 11. 12. 13. 14.

LANGUAGE: English

AB Unsurprisingly, CpG DNA is present in dendritic DNA stimulate a strong innate immune response. There is evidence that DNA-dependent protein kinase (DNA-PK) mediates CpG signaling. Specifically, wortmannin (an inhibitor of phosphatidylinositol 3 kinase [PI3]-kinases including DNA-PK) interferes with CpG-dependent cell activation, and DNA-PK knockout (KO) mice fail to respond to CpG stimulation. Current studies establish that wortmannin actually inhibits the uptake and colocalization of CpG DNA with toll-like receptor (TLR)-9 in endocytic vesicles, thereby preventing CpG-induced activation of the **NF- $\kappa$ B** signaling cascade. We find that DNA-PK is not involved in this process, since three strains of DNA-PK KO mice responded normally to CpG DNA. These results support a model in which CpG signaling is not dependent on TLR-9 but not DNA-PK, and suggest that wortmannin-sensitive members of the PI3-kinase family (e.g., PI3K, but not DNA-PK) are involved in TLR-9.

REFERENCE COUNT: 2. THERE ARE 2 OTHER REFERENCES AVAILABLE FOR THIS  
 REFERENCE COUNT. THERE IS NO AVAILABLE IN THE REFERENCE

As a result, the model is able to capture the nonlinear relationship between the variables and the response variable, and it is able to handle the non-normal distribution of the response variable. The model is able to capture the nonlinear relationship between the variables and the response variable, and it is able to handle the non-normal distribution of the response variable.

INVENTOR:  BY: 

PATENT AND FILE NO. : 2011/0000000 Filed by Inventor(s)

**CONCLUSIONS:** The results of this study suggest that the use of a single, standardized, and validated questionnaire is a feasible and reliable method for assessing the prevalence of self-reported SLE in a community-based sample of the adult population of a large, urban, multiethnic, and multi-racial city.

1.  $\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$  (Probability of getting two heads)  
 2.  $\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$  (Probability of getting two tails)  
 3.  $\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$  (Probability of getting one head and one tail)  
 4.  $\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$  (Probability of getting one tail and one head)

<sup>a</sup> The number of subjects who were included in each group was 10.

FAMILY ACC. 272. 19. 1900. 1.

PATENT INFORMATION:

[illegible]



2-001232

R: `AD, BE, CG, DE, EF, FG, GH, HI, IK, IL, JM, KN, LP, MQ, NR, OS, PT, QV, RW, SX, TY, UZ, VZ, WZ, XX, YY, ZZ`  
 R: `AD, BE, CG, DE, EF, FG, GH, HI, IK, IL, JM, KN, LP, MQ, NR, OS, PT, QV, RW, SX, TY, UZ, VZ, WZ, XX, YY, ZZ`

BN	1949-10-01C	A	19491224
W	2000-10-01A	W	20001222

Activation of the NF- $\kappa$ B response by NF- $\kappa$ B inducers, induction of an energetic response by NF- $\kappa$ B inhibitors, and the inhibition and activation of immune response by the administration of an activator or inhibitor of NF- $\kappa$ B is disclosed. Examples of NF- $\kappa$ B inhibitors include I. $\kappa$ B.alpha., PSI, a nucleotide sequence encoding I. $\kappa$ B.alpha., anti-sense nucleic acid encoding an NF- $\kappa$ B sequence, such as Rel B, and anti-NF- $\kappa$ B antibodies. Examples of NF- $\kappa$ B inducers include NIK, MEKK, IKK $\alpha$ , TIRFEE and Rel B. Also disclosed are vectors encoding inducers and inhibitors of NF- $\kappa$ B, for example adenoviral vectors.

11. 12. 13. 14. 15. 16. 17. 18. 19. 20. 21. 22. 23. 24. 25. 26. 27. 28. 29. 30. 31. 32. 33. 34. 35. 36. 37. 38. 39. 40. 41. 42. 43. 44. 45. 46. 47. 48. 49. 50. 51. 52. 53. 54. 55. 56. 57. 58. 59. 60. 61. 62. 63. 64. 65. 66. 67. 68. 69. 70. 71. 72. 73. 74. 75. 76. 77. 78. 79. 80. 81. 82. 83. 84. 85. 86. 87. 88. 89. 90. 91. 92. 93. 94. 95. 96. 97. 98. 99. 100. 101. 102. 103. 104. 105. 106. 107. 108. 109. 110. 111. 112. 113. 114. 115. 116. 117. 118. 119. 120. 121. 122. 123. 124. 125. 126. 127. 128. 129. 130. 131. 132. 133. 134. 135. 136. 137. 138. 139. 140. 141. 142. 143. 144. 145. 146. 147. 148. 149. 150. 151. 152. 153. 154. 155. 156. 157. 158. 159. 160. 161. 162. 163. 164. 165. 166. 167. 168. 169. 170. 171. 172. 173. 174. 175. 176. 177. 178. 179. 180. 181. 182. 183. 184. 185. 186. 187. 188. 189. 190. 191. 192. 193. 194. 195. 196. 197. 198. 199. 200. 201. 202. 203. 204. 205. 206. 207. 208. 209. 210. 211. 212. 213. 214. 215. 216. 217. 218. 219. 220. 221. 222. 223. 224. 225. 226. 227. 228. 229. 230. 231. 232. 233. 234. 235. 236. 237. 238. 239. 240. 241. 242. 243. 244. 245. 246. 247. 248. 249. 250. 251. 252. 253. 254. 255. 256. 257. 258. 259. 260. 261. 262. 263. 264. 265. 266. 267. 268. 269. 270. 271. 272. 273. 274. 275. 276. 277. 278. 279. 280. 281. 282. 283. 284. 285. 286. 287. 288. 289. 290. 291. 292. 293. 294. 295. 296. 297. 298. 299. 300. 301. 302. 303. 304. 305. 306. 307. 308. 309. 310. 311. 312. 313. 314. 315. 316. 317. 318. 319. 320. 321. 322. 323. 324. 325. 326. 327. 328. 329. 330. 331. 332. 333. 334. 335. 336. 337. 338. 339. 340. 341. 342. 343. 344. 345. 346. 347. 348. 349. 350. 351. 352. 353. 354. 355. 356. 357. 358. 359. 360. 361. 362. 363. 364. 365. 366. 367. 368. 369. 370. 371. 372. 373. 374. 375. 376. 377. 378. 379. 380. 381. 382. 383. 384. 385. 386. 387. 388. 389. 390. 391. 392. 393. 394. 395. 396. 397. 398. 399. 400. 401. 402. 403. 404. 405. 406. 407. 408. 409. 410. 411. 412. 413. 414. 415. 416. 417. 418. 419. 420. 421. 422. 423. 424. 425. 426. 427. 428. 429. 430. 431. 432. 433. 434. 435. 436. 437. 438. 439. 440. 441. 442. 443. 444. 445. 446. 447. 448. 449. 450. 451. 452. 453. 454. 455. 456. 457. 458. 459. 460. 461. 462. 463. 464. 465. 466. 467. 468. 469. 470. 471. 472. 473. 474. 475. 476. 477. 478. 479. 480. 481. 482. 483. 484. 485. 486. 487. 488. 489. 490. 491. 492. 493. 494. 495. 496. 497. 498. 499. 500. 501. 502. 503. 504. 505. 506. 507. 508. 509. 510. 511. 512. 513. 514. 515. 516. 517. 518. 519. 520. 521. 522. 523. 524. 525. 526. 527. 528. 529. 530. 531. 532. 533. 534. 535. 536. 537. 538. 539. 540. 541. 542. 543. 544. 545. 546. 547. 548. 549. 550. 551. 552. 553. 554. 555. 556. 557. 558. 559. 560. 561. 562. 563. 564. 565. 566. 567. 568. 569. 570. 571. 572. 573. 574. 575. 576. 577. 578. 579. 580. 581. 582. 583. 584. 585. 586. 587. 588. 589. 590. 591. 592. 593. 594. 595. 596. 597. 598. 599. 600. 601. 602. 603. 604. 605. 606. 607. 608. 609. 610. 611. 612. 613. 614. 615. 616. 617. 618. 619. 620. 621. 622. 623. 624. 625. 626. 627. 628. 629. 630. 631. 632. 633. 634. 635. 636. 637. 638. 639. 640. 641. 642. 643. 644. 645. 646. 647. 648. 649. 650. 651. 652. 653. 654. 655. 656. 657. 658. 659. 660. 661. 662. 663. 664. 665. 666. 667. 668. 669. 670. 671. 672. 673. 674. 675. 676. 677. 678. 679. 680. 681. 682. 683. 684. 685. 686. 687. 688. 689. 690. 691. 692. 693. 694. 695. 696. 697. 698. 699. 700. 701. 702. 703. 704. 705. 706. 707. 708. 709. 710. 711. 712. 713. 714. 715. 716. 717. 718. 719. 720. 721. 722. 723. 724. 725. 726. 727. 728. 729. 730. 731. 732. 733. 734. 735. 736. 737. 738. 739. 740. 741. 742. 743. 744. 745. 746. 747. 748. 749. 750. 751. 752. 753. 754. 755. 756. 757. 758. 759. 760. 761. 762. 763. 764. 765. 766. 767. 768. 769. 770. 771. 772. 773. 774. 775. 776. 777. 778. 779. 780. 781. 782. 783. 784. 785. 786. 787. 788. 789. 790. 791. 792. 793. 794. 795. 796. 797. 798. 799. 800. 801. 802. 803. 804. 805. 806. 807. 808. 809. 810. 811. 812. 813. 814. 815. 816. 817. 818. 819. 820. 821. 822. 823. 824. 825. 826. 827. 828. 829. 830. 831. 832. 833. 834. 835. 836. 837. 838. 839. 840. 841. 842. 843. 844. 845. 846.

1.  $\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$

INVENTOR(S): Chen, Alan; Elvashoff, Ellen; Fong, Sherman; Goddard, Arney; J. J. Keel, Paul J.; Grimaldi, Christopher J.; Linney, Austin L.; Li, Huanhong; Hillan, Kenneth J.; Jones, Daniel; Van, Lockeren Menno; Vandien, Richard J.; Watanabe, Shinji K.; Williams, P. Mickey; Wood, William J.; Yashima, Etsuo S.

SOURCE: FBI LAB. REF. # 15-10

DOCUMENT TYPE: Patent

FAMILY ACC. NUM. COUNT: 27

TABLE 1. *Continued*

| 2019 |   |   |   |   |   |   |   |   |    | 2020 |    |    |    |    |    |    |    |    |    | 2021 |    |    |    |    |    |    |    |    |    | 2022 |    |    |    |    |    |    |    |    |    | 2023 |    |    |    |    |    |    |    |    |    | 2024 |    |    |    |    |    |    |    |    |    | 2025 |    |    |    |    |    |    |    |    |    | 2026 |    |    |    |    |    |    |    |    |    | 2027 |    |    |    |    |    |    |    |    |    | 2028 |    |    |    |    |    |    |    |    |     | 2029 |  |  |  |  |  |  |  |  |  | 2030 |  |  |  |  |  |  |  |  |  |
|------|---|---|---|---|---|---|---|---|----|------|----|----|----|----|----|----|----|----|----|------|----|----|----|----|----|----|----|----|----|------|----|----|----|----|----|----|----|----|----|------|----|----|----|----|----|----|----|----|----|------|----|----|----|----|----|----|----|----|----|------|----|----|----|----|----|----|----|----|----|------|----|----|----|----|----|----|----|----|----|------|----|----|----|----|----|----|----|----|----|------|----|----|----|----|----|----|----|----|-----|------|--|--|--|--|--|--|--|--|--|------|--|--|--|--|--|--|--|--|--|
| 2019 |   |   |   |   |   |   |   |   |    | 2020 |    |    |    |    |    |    |    |    |    | 2021 |    |    |    |    |    |    |    |    |    | 2022 |    |    |    |    |    |    |    |    |    | 2023 |    |    |    |    |    |    |    |    |    | 2024 |    |    |    |    |    |    |    |    |    | 2025 |    |    |    |    |    |    |    |    |    | 2026 |    |    |    |    |    |    |    |    |    | 2027 |    |    |    |    |    |    |    |    |    | 2028 |    |    |    |    |    |    |    |    |     | 2029 |  |  |  |  |  |  |  |  |  | 2030 |  |  |  |  |  |  |  |  |  |
| 1    | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11   | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21   | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31   | 32 | 33 | 34 | 35 | 36 | 37 | 38 | 39 | 40 | 41   | 42 | 43 | 44 | 45 | 46 | 47 | 48 | 49 | 50 | 51   | 52 | 53 | 54 | 55 | 56 | 57 | 58 | 59 | 60 | 61   | 62 | 63 | 64 | 65 | 66 | 67 | 68 | 69 | 70 | 71   | 72 | 73 | 74 | 75 | 76 | 77 | 78 | 79 | 80 | 81   | 82 | 83 | 84 | 85 | 86 | 87 | 88 | 89 | 90 | 91   | 92 | 93 | 94 | 95 | 96 | 97 | 98 | 99 | 100 |      |  |  |  |  |  |  |  |  |  |      |  |  |  |  |  |  |  |  |  |
| 1    | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11   | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21   | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31   | 32 | 33 | 34 | 35 | 36 | 37 | 38 | 39 | 40 | 41   | 42 | 43 | 44 | 45 | 46 | 47 | 48 | 49 | 50 | 51   | 52 | 53 | 54 | 55 | 56 | 57 | 58 | 59 | 60 | 61   | 62 | 63 | 64 | 65 | 66 | 67 | 68 | 69 | 70 | 71   | 72 | 73 | 74 | 75 | 76 | 77 | 78 | 79 | 80 | 81   | 82 | 83 | 84 | 85 | 86 | 87 | 88 | 89 | 90 | 91   | 92 | 93 | 94 | 95 | 96 | 97 | 98 | 99 | 100 |      |  |  |  |  |  |  |  |  |  |      |  |  |  |  |  |  |  |  |  |

[illegible]



[illegible]

| Case | Age | Sex | Site    | Pathologic     | Survival |
|------|-----|-----|---------|----------------|----------|
| 1    | 50  | F   | Stomach | Adenocarcinoma | 10 mo    |
| 2    | 55  | M   | Stomach | Adenocarcinoma | 12 mo    |
| 3    | 60  | F   | Stomach | Adenocarcinoma | 15 mo    |
| 4    | 65  | M   | Stomach | Adenocarcinoma | 18 mo    |
| 5    | 70  | F   | Stomach | Adenocarcinoma | 20 mo    |
| 6    | 75  | M   | Stomach | Adenocarcinoma | 22 mo    |
| 7    | 80  | F   | Stomach | Adenocarcinoma | 25 mo    |
| 8    | 85  | M   | Stomach | Adenocarcinoma | 28 mo    |
| 9    | 90  | F   | Stomach | Adenocarcinoma | 30 mo    |
| 10   | 95  | M   | Stomach | Adenocarcinoma | 32 mo    |
| 11   | 100 | F   | Stomach | Adenocarcinoma | 35 mo    |
| 12   | 105 | M   | Stomach | Adenocarcinoma | 38 mo    |
| 13   | 110 | F   | Stomach | Adenocarcinoma | 40 mo    |
| 14   | 115 | M   | Stomach | Adenocarcinoma | 42 mo    |
| 15   | 120 | F   | Stomach | Adenocarcinoma | 45 mo    |
| 16   | 125 | M   | Stomach | Adenocarcinoma | 48 mo    |
| 17   | 130 | F   | Stomach | Adenocarcinoma | 50 mo    |
| 18   | 135 | M   | Stomach | Adenocarcinoma | 52 mo    |
| 19   | 140 | F   | Stomach | Adenocarcinoma | 55 mo    |
| 20   | 145 | M   | Stomach | Adenocarcinoma | 58 mo    |
| 21   | 150 | F   | Stomach | Adenocarcinoma | 60 mo    |
| 22   | 155 | M   | Stomach | Adenocarcinoma | 62 mo    |
| 23   | 160 | F   | Stomach | Adenocarcinoma | 65 mo    |
| 24   | 165 | M   | Stomach | Adenocarcinoma | 68 mo    |
| 25   | 170 | F   | Stomach | Adenocarcinoma | 70 mo    |
| 26   | 175 | M   | Stomach | Adenocarcinoma | 72 mo    |
| 27   | 180 | F   | Stomach | Adenocarcinoma | 75 mo    |
| 28   | 185 | M   | Stomach | Adenocarcinoma | 78 mo    |
| 29   | 190 | F   | Stomach | Adenocarcinoma | 80 mo    |
| 30   | 195 | M   | Stomach | Adenocarcinoma | 82 mo    |
| 31   | 200 | F   | Stomach | Adenocarcinoma | 85 mo    |
| 32   | 205 | M   | Stomach | Adenocarcinoma | 88 mo    |
| 33   | 210 | F   | Stomach | Adenocarcinoma | 90 mo    |
| 34   | 215 | M   | Stomach | Adenocarcinoma | 92 mo    |
| 35   | 220 | F   | Stomach | Adenocarcinoma | 95 mo    |
| 36   | 225 | M   | Stomach | Adenocarcinoma | 98 mo    |
| 37   | 230 | F   | Stomach | Adenocarcinoma | 100 mo   |
| 38   | 235 | M   | Stomach | Adenocarcinoma | 102 mo   |
| 39   | 240 | F   | Stomach | Adenocarcinoma | 105 mo   |
| 40   | 245 | M   | Stomach | Adenocarcinoma | 108 mo   |
| 41   | 250 | F   | Stomach | Adenocarcinoma | 110 mo   |
| 42   | 255 | M   | Stomach | Adenocarcinoma | 112 mo   |
| 43   | 260 | F   | Stomach | Adenocarcinoma | 115 mo   |
| 44   | 265 | M   | Stomach | Adenocarcinoma | 118 mo   |
| 45   | 270 | F   | Stomach | Adenocarcinoma | 120 mo   |
| 46   | 275 | M   | Stomach | Adenocarcinoma | 122 mo   |
| 47   | 280 | F   | Stomach | Adenocarcinoma | 125 mo   |
| 48   | 285 | M   | Stomach | Adenocarcinoma | 128 mo   |
| 49   | 290 | F   | Stomach | Adenocarcinoma | 130 mo   |
| 50   | 295 | M   | Stomach | Adenocarcinoma | 132 mo   |
| 51   | 300 | F   | Stomach | Adenocarcinoma | 135 mo   |
| 52   | 305 | M   | Stomach | Adenocarcinoma | 138 mo   |
| 53   | 310 | F   | Stomach | Adenocarcinoma | 140 mo   |
| 54   | 315 | M   | Stomach | Adenocarcinoma | 142 mo   |
| 55   | 320 | F   | Stomach | Adenocarcinoma | 145 mo   |
| 56   | 325 | M   | Stomach | Adenocarcinoma | 148 mo   |
| 57   | 330 | F   | Stomach | Adenocarcinoma | 150 mo   |
| 58   | 335 | M   | Stomach | Adenocarcinoma | 152 mo   |
| 59   | 340 | F   | Stomach | Adenocarcinoma | 155 mo   |
| 60   | 345 | M   | Stomach | Adenocarcinoma | 158 mo   |
| 61   | 350 | F   | Stomach | Adenocarcinoma | 160 mo   |
| 62   | 355 | M   | Stomach | Adenocarcinoma | 162 mo   |
| 63   | 360 | F   | Stomach | Adenocarcinoma | 165 mo   |
| 64   | 365 | M   | Stomach | Adenocarcinoma | 168 mo   |
| 65   | 370 | F   | Stomach | Adenocarcinoma | 170 mo   |
| 66   | 375 | M   | Stomach | Adenocarcinoma | 172 mo   |
| 67   | 380 | F   | Stomach | Adenocarcinoma | 175 mo   |
| 68   | 385 | M   | Stomach | Adenocarcinoma | 178 mo   |
| 69   | 390 | F   | Stomach | Adenocarcinoma | 180 mo   |
| 70   | 395 | M   | Stomach | Adenocarcinoma | 182 mo   |
| 71   | 400 | F   | Stomach | Adenocarcinoma | 185 mo   |
| 72   | 405 | M   | Stomach | Adenocarcinoma | 188 mo   |
| 73   | 410 | F   | Stomach | Adenocarcinoma | 190 mo   |
| 74   | 415 | M   | Stomach | Adenocarcinoma | 192 mo   |
| 75   | 420 | F   | Stomach | Adenocarcinoma | 195 mo   |
| 76   | 425 | M   | Stomach | Adenocarcinoma |          |







DOCUMENT TYPE: Biology  
LANGUAGE: English

AB **Dendritic cells** Dendritic cells present a unique phenotype and function in the immune system. To identify genes that are up-regulated in DCs, we used a cDNA microarray and a complementary DNA (cDNA) library. We identified 100 genes that were up-regulated in DCs. Among these genes, 40 genes showed expression changes at the RNA level using oligonucleotide array complementary to 100 human genes. About 40% of the genes were expressed in DCs. A total of 15 genes were regulated during DC differentiation or maturation. Most of these genes were not previously associated with DCs and include genes encoding secreted proteins as well as genes involved in cell adhesion, signaling, and lipid metabolism. Protein analysis of the same cell populations was done using two-dimensional gel electrophoresis. A total of 900 distinct protein spots were included, and 4% of them exhibited quantitative changes during DC differentiation and maturation. Differentially expressed proteins were identified by mass spectrometry and found to represent proteins with Ca<sup>2+</sup> binding, fatty acid binding, or chaperone activities as well as proteins involved in cell motility. In addition, proteomic analysis provided an assessment of post-translational modifications. The chaperone protein, calnexin, was found to undergo cleavage, yielding a novel form. The combined oligonucleotide microarray and proteomic approaches have demonstrated novel genes associated with DC differentiation and maturation and have allowed analysis of post-translational modifications of specific proteins as part of these processes.

REFERENCE COUNT: 1 (MORE AVAILABLE WITH REFERENCES AVAILABLE FOR THIS REFERENCE. ALL CITATIONS AVAILABLE IN THE RE FORMAT)

121 **AMINO ACID SEQUENCE OF THE PROTEIN**  
ACCESSION NUMBER: P01344  
DOCUMENT NUMBER: 1316100  
TITLE: A toll-like receptor recognizes bacterial DNA.  
[Erratum to document cited in CA134:161862]  
AUTHOR(S): Hemmi, Hiroaki; Takeuchi, Osamu; Kawai, Taro; Kaisho, Tsuneyasu; Sato, Shintaro; Sanjo, Hideaki; Matsumoto, Makoto; Hoshino, Katsunaki; Wagner, Hermann; Takeda, Kiyoshi; Akira, Shizuo  
CORPORATE SOURCE: Department of Host Defense, Research Institute for Microbial Diseases, Osaka University and The Center for Environmental Science and Technology, Osaka, Japan, 565-0871, Japan  
SOURCE: Nature Immunology, Vol. 1, No. 12, 1999, 1131-1135  
PUBLISHER: Nature Publishing Group  
JOURNAL TITLE: Nature Immunology  
ISSN: 1474-2675  
AB The toll-like receptor (TLR) family is a class of pattern recognition receptors that recognize conserved molecular patterns of pathogens. Here, we show that the TLR1/2 heterodimer recognizes a conserved motif of lipopeptides derived from Gram-negative bacteria. This motif is recognized by the TLR1/2 heterodimer in a sequence-specific manner. The TLR1/2 heterodimer is a member of the TLR family and is involved in the recognition of bacterial DNA.

122 **AMINO ACID SEQUENCE OF THE PROTEIN**  
ACCESSION NUMBER: P01344  
DOCUMENT NUMBER: 1316100  
TITLE: A toll-like receptor recognizes bacterial DNA.  
AUTHOR(S): Hemmi, Hiroaki; Takeuchi, Osamu; Kawai, Taro; Kaisho, Tsuneyasu; Sato, Shintaro; Sanjo, Hideaki; Matsumoto, Makoto; Hoshino, Katsunaki; Wagner, Hermann; Takeda, Kiyoshi; Akira, Shizuo  
CORPORATE SOURCE: Department of Host Defense, Research Institute for Microbial Diseases, Osaka University and The Center for Environmental Science and Technology, Osaka, Japan, 565-0871, Japan  
SOURCE: Nature Immunology, Vol. 1, No. 12, 1999, 1131-1135  
PUBLISHER: Nature Publishing Group  
JOURNAL TITLE: Nature Immunology  
ISSN: 1474-2675

AP DNA from bacteria is a strongly immunogenic in mammalian immune cells, which depend on the presence of unmethylated CpG dinucleotides in the bacterial DNA. In contrast, mammalian DNA has a low frequency of CpG dinucleotides, and these are mostly methylated; therefore, mammalian DNA does not have immuno-stimulatory activity. CpG DNA induces a strong T-helper-1-like inflammatory response. Accumulating evidence has revealed the therapeutic potential of CpG DNA as adjuvants for vaccination strategies for cancer, allergy and infectious diseases. Despite its potential utility, the molecular mechanism by which CpG DNA activates immune cells remains unclear. Here the authors provide a detailed analysis of CpG DNA as a stimulatory TLR in dendritic cells. They show that CpG DNA is a stimulatory TLR in dendritic cells, which elicits a strong T-helper-1-like response and show any response to CpG DNA, including activation of dendritic cells, inflammatory cytokine production, and expression of costimulatory molecules in **dendritic cells**. TLR9-/- mice showed a significant reduction in the local effect of CpG DNA without any effect on the systemic pro-inflammatory cytokine levels. The induction of T-helper-1 and T-helper-type-1 response was also abolished in TLR9-/- mice. Thus, vertebrate immune systems appear to have evolved a specialized TLR-like receptor that distinguishes bacterial DNA from self-DNA.

L21 ANSWER 15 OF 15 ENBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

TITLE: Preexposure of marine macrophages to CpG-containing oligonucleotides results in nuclear factor- $\kappa$ B-dependent interleukin-6 transcriptional activation.

Figure 1. The effect of the concentration of the *Agrobacterium* suspension on the transformation efficiency of *Agrobacterium* strains. The concentration of the *Agrobacterium* suspension was 10<sup>6</sup> cells/ml (○), 10<sup>7</sup> cells/ml (□), 10<sup>8</sup> cells/ml (△), and 10<sup>9</sup> cells/ml (◇). The data were the mean of three independent experiments. Error bars represent standard deviation.

LANGUAGE: English

AB Background: CpG-containing oligonucleotides (CpG ODN) is associated with immunomodulation of the innate immune response. Pre-exposure of macrophages to CpG ODN elicits a hyper-responsiveness to subsequent lipopolysaccharide (LPS) stimulation. We tested the hypothesis that this effect is due to decreased nuclear translocation of nuclear factor- $\kappa$ B (NF- $\kappa$ B). Methods: Murine macrophage-like RAW 264.7 cells were incubated with 1.0  $\mu$ g/mL CpG-containing oligonucleotides (CpG ODN) for 24 hours followed by restimulation with 1.0  $\mu$ g/mL LPS for 15 minutes. The cells were then treated with an NF- $\kappa$ B sensitive inhibitor, Bay K 86.96, and a NF- $\kappa$ B inhibitor, Bay 11-7082. Cytoplasmic and nuclear extracts were analyzed for NF- $\kappa$ B. Results: CpG ODN treatment of RAW 264.7 cells increased NF- $\kappa$ B activity, as measured by NF- $\kappa$ B reporter gene activity, and increased NF- $\kappa$ B nuclear translocation. NF- $\kappa$ B activity and nuclear translocation were inhibited by Bay K 86.96 and Bay 11-7082. Conclusion: CpG ODN treatment of macrophages increases NF- $\kappa$ B activity and nuclear translocation, which is inhibited by NF- $\kappa$ B inhibitors. This suggests that CpG ODN treatment of macrophages may increase NF- $\kappa$ B activity and nuclear translocation, which is inhibited by NF- $\kappa$ B inhibitors. This suggests that CpG ODN treatment of macrophages may increase NF- $\kappa$ B activity and nuclear translocation, which is inhibited by NF- $\kappa$ B inhibitors.



expression of the inhibitory  $\gamma$  NF- $\kappa$ B  
homodimer and decreased NF- $\kappa$ B activity  
without inhibition of NF- $\kappa$ B kinase. NF- $\kappa$ B activity is also  
activity of the NF- $\kappa$ B complex. These results suggest that the  
distribution of the NF- $\kappa$ B complex is not uniform in all cells  
in the same tissue.

L233 ANSWER : 1  
 ACCESSION NUMBER: 1  
 DOCUMENT NUMBER: 1  
 TITLE: **Endothelial and dendritic cells in**  
**transplantation and autoimmune**  
**disease**  
 AUTHOR : **Chen, Y ; Thomas, Angus W.**  
 CORPORATE SOURCE: **Johns Hopkins Transplantation Institute, University**  
**of Maryland Medical Center, 200 Lothrop Street, E1554,**  
**Biomedical Science Tower, Pittsburgh, PA, 15213:**  
**l23max.upmc.edu USA**  
 SOURCE: **Transplantation (Baltimore), (January 15, 2002) Vol. 73,**  
**No. 1 Supplement , pp. S19-S22.**  
**<http://www.transplantjournal.com/>. print.**  
**ISSN: G041-1337.**  
 DOCUMENT TYPE: **General Review**  
 LANGUAGE: **English**

AB Dendritic cells (DC) constitute a complex system of uniquely well-equipped antigen-presenting cells that initiate and regulate immune responses. Extensive recent studies have improved our understanding of DC development, differentiation, activation, and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and functional capacity. These factors seem to determine the T-cell activating stimulus and type of T cell response—T helper 1, T helper 2, or T regulatory—induced by DC (1). Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T-regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas (CD95) ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of gene transcription regulatory proteins (e.g., nuclear factor-kappaB) can impart **tolerogenic** potential to DC (2). Manipulation of DC by control of their maturation and differentiation, or genetic engineering of these cells to express immunosuppressive molecules, offers potential for therapy of allograft rejection and autoimmune disease. In this brief review, we outline principles and methods for generation of "tolerogenic" DC and outcomes that have been reported in experimental models. A brief concluding comment is provided at the end.

[illegible]

[illegible]

L25 ACCESSION NUMBER: 0198-8859(199706)62:10;1-L  
ACCESSION NUMBER: 0198-8859(199706)62:10;1-L  
DOCUMENT NUMBER: 0198-8859(199706)62:10;1-L  
TITLE: Altering mRNA microarray profiles of  
**tolerogenic dendritic cells**  
AUTHOR(S): Suda-Kohda, Corvesini, N.; Piazza, F.; Ho, E.;  
Mickelson, E.; LeNecrot, J.; Dalla-Favera, R.;  
Corvesini, K.  
CORPORATE SOURCE: Department of Pathology, Columbia University, New  
York, NY, USA  
SOURCE: Human Immunology (2001), 62(10), 1069-1072  
CODEN: HUIMDQ; ISSN: 0198-8859  
PUBLISHER: Elsevier Science Inc.  
DOCUMENT TYPE: Journal  
LANGUAGE: English

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2002:199192 CAPLUS  
DOCUMENT NUMBER: 137:41416  
TITLE: Prolongation of cardiac allograft survival using dendritic cells treated with NF-kappa-B decoy oligodeoxynucleotides  
AUTHOR(S): Mann, Karin; Li, Hui; Bunn, J. Andrew; Chan, Anthony S.; Chen, Zhongqiang; Dandekar, Pradyumn K.; Li, Wei; Lippert, John W.; Park, Joon Y.; Rostoff, Eyal; Wang, Jie  
CORPORATE SOURCE: Department of Cell Biology, Biochemistry and Biophysics, Johns Hopkins University, Baltimore, MD, 12061, USA  
SOURCE: Journal of Cellular Therapy and Transplantation, Vol. 1, No. 4, 431-437  
ISSN: 1547-3847; ISSN: 1547-3847  
PUBLISHED: 2002-01-01  
DOCUMENT TYPE: Journal Article  
LANGUAGE: English

AB Dendritic cells (DCs) play a key role in immune response but are also manipulated by tumor cells to suppress antitumor immunity. The expression of transcription factors, including NF- $\kappa$ B, IRF3, IRF7, and others, in DCs correlates with their capacity to induce or suppress immune responses. Expression of these genes is associated with NF- $\kappa$ B-dependent transcription. In this regard, the **tolerogenicity** has been linked with impaired NF- $\kappa$ B-dependent transcription of transcriptionary genes as well as NF- $\kappa$ B translocation to the nucleus. In this regard, we demonstrate that double-stranded DNA (dsDNA) and the viral protein cGAS induce NF- $\kappa$ B. NF- $\kappa$ B and cGAS are essential for the induction of IRF3-IRF7-dependent transcription of IRF3-IRF7 target genes.

transcription of the reporter gene. Moreover, exposure of DC to the cleavable polyacrylate inhibited lipopolysaccharide (LPS)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF- $\kappa$ B JEN selectively suppressed the cell-surface expression of costimulatory moles. without interfering with MHC class I or class II expression. Furthermore, NF- $\kappa$ B JEN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was assocd. with inhibition of TH1-type cytokine prodn. Finally, infusion of NF- $\kappa$ B JEN-mobilized bone marrow-derived DC in allogeneic recipients prior to heart transplant with resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF- $\kappa$ B and other transcriptional pathways involved in immune stimulation in DC using JEN may provide a novel approach to promote tolerance induction in organ transplantation. J. Clin. Invest. 105:1111-1120, 2000.

REFERENCE COUNT: 1 (1999) APP. 14 (1999) REFERRED BY AVAILABLE FOR THIS  
AP. 14. ALL CLAIMS IN AVAILABLE IN THE RE FORMAI

LBS ANKERS : 0  
ACCESSION NUMBER: 87-9630  
DOCUMENT NUMBER: 87-9630  
TITLE:  
**Hepatitis B virus-induced liver graft survival by dendritic cells preferentially engineered with NF-kappa B**  
**cytokines expressed from adenoviral vectors containing CTLA4-Ig.**  
AUTHOR: Fanbin F Andrew; Peng Liansha; Liang Xiaoyan; Chen Zengyou;  
Wang Huanru; Ma Linlin; Backstein Holger; Robbins Paul D;  
Thomson Angus W; Pang John J; Qian Shiguang; Lu Lina  
CORPORATE SOURCE: Department of Surgery and Thomas E. Starzl Transplantation Institute,  
University of Pittsburgh Medical Center,  
University of Pittsburgh, Pittsburgh, PA 15213, USA.  
CONTRACT NUMBER: AI41011 (NIAID)  
SOURCE: JOURNAL OF IMMUNOLOGY, [2004 Sep; 163(3) 3332-41.  
Journal code: IMEDITEB. ISSN: 0256-7608].  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal Article; JOURNAL ARTICLE  
LANGUAGE: English  
FILE SEGMENT: Abstract Index Medicus Journals; Priority Journals  
ENTRY MONTH:  
ENTRY YEAR:

Adenoviral-transduced dendritic cells (DCs) can be genetically engineered using adenoviral (Ad) vectors to express immunosuppressive molecules that promote T cell unresponsiveness. The success of these DCs for therapy of allograft rejection has been limited in part by the potential of the adenovirus to promote DC maturation and the inherent ability of the DC to undergo maturation following in vivo administration. DC maturation occurs via NF-kappaB-dependent mechanisms, which can be blocked by fully immunostimulated "decoy" oligodeoxynucleotides (ODNs) containing binding sites for NF-kappaB. Herein, we describe the combined use of NF-kappaB ODNs and rAd vectors encoding CTLA4-Ig (Ad CTLA4-Ig) to generate stably immature murine myeloid DCs that secrete the potent costimulation blocking agent. These Ad CTLA4-Ig-transduced ODN DCs exhibit markedly impaired allostimulatory ability and promote adoptive activated T cells. Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL/6; B10.H-2K<sup>b</sup>) before transplant significantly prolongs MHC-mismatched (B10.H-2K<sup>b</sup> x B10.H-2K<sup>d</sup>) heart allograft survival in B10.H-2K<sup>b</sup> recipients. These results suggest that the combination of Ad CTLA4-Ig and ODNs may be useful to promote **tolerogenicity**, and that the use of Ad CTLA4-Ig-transduced ODN DCs as donor cells, and not as stimulators, may be useful in the treatment of allograft rejection. The use of NF-kappaB antisense oligodeoxynucleotides with the encoding a potent costimulation blocking agent may provide a therapy of allograft rejection or other immune-mediated pathologic conditions associated with systemic immunosuppression.

155 ALPHABETICALLY: MEDICINE DUPLICATE 2  
ACCESSION NUMBER: 11-10000 MEDICINE  
DOCUMENT NUMBER: 11-10000  
TITLE: Manipulation of dendritic cells for  
tolerance induction in transplantation and autoimmune  
disease.  
AUTHOR: Lu Lirag; Thomas E. Starzl  
CORPORATE SOURCE: Thomas E. Starzl Transplantation Institute, Department of  
Surgery, University of Pittsburgh Medical Center,  
Pittsburgh, Pennsylvania 15261, USA. Email: starzl@upmc.edu  
CONTRACT NUMBER:  
DEPT. FILE  
SPONS. FILE  
SOURCE: 11-10000

PUB. COUNTRY:  
DOCUMENT TYPE:

LANGUAGE:  
FILE SEGMENT:  
ENTRY MONTH:  
ENTRY DATE:

55

125    AN UNP    100  
ACCESS: 11 NUMBER:  
DOCUMENT NUMBER:  
TITLE:

## APPENDIX 3

1. *Journal of the American Medical Association*, 1997; 277: 103-107.

AUTHOR :

CONTRACT NUMBER:  
SOURCE:

FILE NO. :  
 ENTRY NO. :  
 FILE NO. :  
 ENTRY NO. :  
 FILE NO. :  
 ENTRY NO. :  
 FILE NO. :  
 ENTRY NO. :

22









L15 ANSWER 1 OF 2 TABLE COPYRIGHT 1999 BY AAV

ACCESSION NUMBER:

DOCUMENT NUMBER:

TITLE:

Use of tolerogenic dendritic cells for enhancing tolerogenicity in a host and methods for making the same

AUTHOR(S):

Lin, Lina; Hsiao, Nick; Harnaha, D. Andrew; Qian, Shiguang; Li, Wei; Li, Lina; Harnaha, D. Andrew; Qian, Shiguang; Li, Wei; Li, Lina

CORPORATE SOURCE:

Department of Molecular Genetics and Biochemistry, University of Pittsburgh, Pittsburgh, PA, 15261, USA

SOURCE:

Molecular Therapy (2000), 1(5, Pt. 1), 430-437

CODEN: MTHOK; ISSN: 1525-0016

PUBLISHER:

Academic Press

DOCUMENT TYPE:

Journal

LANGUAGE:

English

AB Dendritic cells (DC) classically promote immune responses but can be manipulated to induce antigen-specific hyporesponsiveness in vitro. The expression of costimulatory moles (CD40, CD86, CD80) at the DC cell surface correlates with their capacity to induce or suppress immune responses. Expression of these moles is associated with NF-kappa-B-dependent transcription of their genes. DC **tolerogenicity** has been associated with impaired NF-kappa-B-dependent transcription of costimulatory genes as well as NF-kappa-B translocation to the nucleus. In this report, we demonstrate that a bone-marrow-derived DC (BM-DC) can be manipulated to enhance tolerogenicity. BM-DCs are efficiently incorporated by bone marrow-derived DC and specifically inhibit NF-kappa-B-dependent transcription of a reporter gene. Moreover, exposure of DC to the oligonucleotide decoys inhibited lipopolysaccharide (LPS)-induced nitric oxide prodn., a marker of DC maturation. Treatment of bone marrow-derived DC progenitors with NF-kappa.B ODN selectively suppressed the cell-surface expression of costimulatory moles without interfering with MHC class I or class II expression. Furthermore, NF-kappa.B ODN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was associated with inhibition of Th1-type cytokine prodn. Finally, infusion of NF-kappa.B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF-kappa.B and other transcriptional pathways involved in immune stimulation in DC using ODN decoy approaches could be one means to promote tolerance induction in solid transplantation. (C) 2000 Academic Press.

REFERENCE(S):

1. THE USE OF TOLERGENIC DENDRITIC CELLS FOR ENHANCING TOLERGENICITY IN A HOST AND METHODS FOR MAKING THE SAME

L15 ANSWER 1 OF 2 TABLE COPYRIGHT 1999 BY AAV

ACCESSION NUMBER:

DOCUMENT NUMBER:

TITLE:

Use of tolerogenic dendritic cells for enhancing tolerogenicity in a host and methods for making the same

INVENTOR(S):

Lin, Lina; Hsiao, Nick; Harnaha, D. Andrew; Qian, Shiguang; Li, Wei; Li, Lina

PATENT ASSIGNER(S):

University of Pittsburgh or the Commonwealth System of Higher Education, USA

SOURCE:

Pat. Appl., 09/011,011

CLASS: B27

DOCUMENT TYPE:

Patent

LANGUAGE:

English

FAMILY APP. NUM. (S): 1

PATENT INFORMATION:

20019423

AB The present invention relates to a tolerogenic mammalian dendritic cell (DC) comprising at least one of the **tolerogenic DCs** of the present invention. In turn, the present invention provides a method for enhancing **tolerogenicity** in a host comprising administering the **tolerogenic** mammalian DCs of the present invention to the host. The **tolerogenic** DCs of the present invention comprise an oligodeoxynucleotide (ODN) which has one or more NF- $\kappa$ B binding sites. The **tolerogenic** DCs of the present invention may further comprise a viral vector, and preferably an adenoviral vector, which does not affect the **tolerogenicity** of the **tolerogenic** DCs when present therein. Enhanced **tolerogenicity** in a host is useful for prolonging foreign graft survival and for treating inflammatory related diseases, such as autoimmune diseases.











inhibitor of NF- $\kappa$ B. The present invention provides a method for the treatment of a host with NF- $\kappa$ B inhibitors of the present invention. The present invention, and particularly an adenoviral vector, which provides for the delivery of the inhibitor to the host is useful for prolonging the host survival and mitigating inflammatory related diseases, such as arthritis diseases.

L14 ANSWER # OF 16 CAPI'S COPYRIGHT 1997

ACCESSION NUMBER: 2001:489042 CAPI'S

DOCUMENT NUMBER: 130:8719

TITLE: Inhibition of glycogen synthase kinase-3 $\beta$  (GSK-3 $\beta$ ) for inhibiting NF- $\kappa$ B, and therapeutic use

INVENTOR(S): Hoefflich, Klaus; Li, Jian; Weigert, Jörn

PATENT ASSIGNEE(S): The Ontario Cancer Institute, Inc.

SOURCE: IGT Int. Appl., No. 97/00000

CLASS: G06F

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY AND INT. NO.: 1

PATENT INFORMATION:

| PATENT NO.  | FILED DATE  | APPLICATION NO. | DATE     |
|---|-------------|-----------------|----------|
| WO 9801047A1  | A1 19970105 | WO 2000-CA1578  | 20001221 |
| R: AT, FR, DE, ES   |             |                 |          |
| EX: AT, BE, CH, CY, DK, IN, ES, FI, FR, GR, GB, IE, IT, LU, MC, NL, PT, SE, TR    |             |                 |          |
| EP 1244451  | A2 19970105 | EP 2000-986951  | 20001221 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR |             |                 |          |
| US 2001053351   | A1 20011220 | US 2001-747552  | 20001221 |
| PRORITY APPLN. INFO.: US 1999-17904P P 19991229                                   |             |                 |          |
| WO 2000-CA1578 W 20001221   |             |                 |          |

AB The activity of NF- $\kappa$ B is modulated through the activity of IKK- $\alpha$ . IKK- $\alpha$  activity. Inhibition or down-regulation of IKK- $\alpha$  results in decreased NF- $\kappa$ B activity. Inappropriate activation of NF- $\kappa$ B has been linked to inflammatory and cancer progression. Inhibition of IKK- $\alpha$  activity by small molecule inhibitors is a potential approach for the treatment or prevention of various diseases. This document provides a method for inhibiting IKK- $\alpha$  activity in cells by using the inhibitor of IKK- $\alpha$  inhibition, which administration to a host with a pro-inflammatory stimulus of TNF receptor-1 ligand, induces selective IKK- $\alpha$  function are also provided.

L14 ANSWER # OF 16 CAPI'S COPYRIGHT 1997

ACCESSION NUMBER: 2001:489042 CAPI'S

DOCUMENT NUMBER: 130:8719

TITLE: Regulation of HIV-1 transcription

AUTHOR(S): Boeruck, Kenneth A.; Safirudin, Mohammed

CORPORATE SOURCE: Department of Immunology Microbiology, Rush Presbyterian St. Luke's Medical Center, Chicago, IL, USA

SOURCE: AIDS Expression Library, No. 97/00000

CLASS: G06F; INTL NO.: 1

PUBLISHED: Published for the first time

DOCUMENT TYPE: Patent

LANGUAGE: English

AB A method for regulating HIV-1 transcription is provided. The method involves the use of a small molecule inhibitor of HIV-1 transcription. The method is useful for the treatment of HIV-1 infection. The method is also useful for the prevention of HIV-1 infection. The method is also useful for the treatment of HIV-1 related diseases. The method is also useful for the prevention of HIV-1 related diseases. The method is also useful for the treatment of HIV-1 related diseases. The method is also useful for the prevention of HIV-1 related diseases.

REFERENCE COUNT: 100 THERE ARE 100 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE REFORMAT.

AB Transcription of the HIV-1 provirus genome is regulated by a complex interplay between viral regulatory proteins and cellular transcription factors that interact with the viral long terminal repeat (LTR) region of HIV-1. However, several cellular transcription factors have been identified that can interact with the HIV-1 LTR; the significance of all of these factors is not clearly understood. In this study the authors have characterized the LTR region of different subtypes of HIV-1 with regard to nucleotide sequence and promoter activity. The LTR region of HIV-1 from peripheral blood mononuclear cells was isolated from individuals originating from 17 different ethnic groups were sequenced and further analyzed for promoter activity. A strong correlation was found between ethnic origin and promoter activity. The authors conclude that the LTR region of HIV-1 may serve as a marker for ethnic origin and suggest that the LTR sequence of the HIV-1 genome may play a role in the ethnic distribution of infection. They also suggest that the LTR region of the HIV-1 genome contains subtype-specific sequences. Interestingly, the authors suggest that the LTR region of the HIV-1 genome may be under selective pressure of the host immune system. Subtypes A, B, C, D, and E are suggested to have potential anti-NF-response elements which could be involved in the regulation of NF response. The authors conclude that the diversity of the LTR may result in HIV-1 strains with different replicative abilities.



ACCESSION NUMBER: 1994:0334- TAYLOR  
 DOCUMENT NUMBER: 1112748  
 TITLE: In vitro study of functional involvement of Sp1, NF-kappa.B/p65, and AP1 in p18-11-myristate-13-acetate-mediated HIV-1 long terminal repeat activation.  
 AUTHOR(S): Li, Yuhli; Nae, Hui; Brannan, Robert E., Jr.  
 CORPORATE SOURCE: Cold Spring Harbor Lab., Cold Spring Harbor, New York, NY, 11724, USA  
 JOURNAL: Journal of Virology, Vol. 68, No. 1, 1994, pp. 111-117.  
 PUBLISHED: 1994, Cold Spring Harbor Laboratory and Cold Spring Harbor Laboratory Press  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB We examd. the cooperative activity between the Sp1 and NF-kappa.B/Rel sites of the human immunodeficiency virus type 1 long terminal repeat in response to phorbol 12-myristate 13-acetate (PMA) stimulation in an in vitro transcription assay. Sp1 sites alone do not account for the activation induced by PMA. When mutations in Sp1 sites were combined with mutations in the NF-kappa.B/Rel sites, a dramatic redn. in PMA-induced transcriptional activity was obsd. This redn. was much greater than the redn. assocd. with mutations involving only the NF-kappa.B/Rel sites. This finding suggests that there is functional cooperation between Sp1 and NF-kappa.B/Rel and that this is one possible mechanism for transcription activation by NF-kappa.B/Rel. The three AP1 sites in the HIV-1 regulatory region of the long terminal repeat, however, seem to be uninvolved in the earliest events of transcriptional activation by PMA.

E14 ANSWER 14: 1994:0334- TAYLOR, RICHARD E., JR.  
 ACCESSION NUMBER: 1994:0334- TAYLOR  
 DOCUMENT NUMBER: 1112748  
 TITLE: Identification of the HIV-1 regulatory human immunodeficiency virus type 1 gene expression.  
 AUTHOR(S): Jordan, D.L. Jr.; Adams, R.; Burkett, Colin A.; Nae, Hui  
 CORPORATE SOURCE: Howard Hughes Medical Institute, University Michigan, Ann Arbor, MI, 48104-3650, USA  
 SOURCE: Journal of Virology, 1994, 68(10), 6820-3  
 CLEN: JOVIAN; ISSN: 0022-538X  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB Human immunodeficiency virus type 1 (HIV-1) gene expression is regulated by an enhancer region composed of multiple potential cis-acting regulatory sites. Here, we describe binding sites for the transcription factor AP1 in the HIV-1 long terminal repeat which mediate HIV enhancer function. One site is embedded within the two previously identified kappa.B elements, and a second site is detected further downstream. Glass 1 is a printing and a graph showing the effect of the AP1 site on the HIV-1 enhancer function. The AP1 site is located between the kappa.B elements. Interestingly, AP1 and NF-kappa.B sites are located in the HIV-1 enhancer region. Mutations in the AP1 site, but not in the NF-kappa.B sites, reduce the HIV-1 enhancer function. The AP1 site is located between the kappa.B elements. The AP1 site is located between the kappa.B elements. The AP1 site is located between the kappa.B elements.

E14 ANSWER 14: 1994:0334- TAYLOR, RICHARD E., JR.  
 ACCESSION NUMBER: 1994:0334- TAYLOR  
 DOCUMENT NUMBER: 1112748  
 TITLE: Identification of the HIV-1 regulatory human immunodeficiency virus type 1 gene expression.  
 AUTHOR(S): Jordan, D.L. Jr.; Adams, R.; Burkett, Colin A.; Nae, Hui  
 CORPORATE SOURCE: Howard Hughes Medical Institute, University Michigan, Ann Arbor, MI, 48104-3650, USA  
 SOURCE: Journal of Virology, 1994, 68(10), 6820-3  
 CLEN: JOVIAN; ISSN: 0022-538X  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English



## PALM INTRANET

Day : Thursday  
Date: 11/21/2002  
Time: 10:36:16

## Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.  
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**Last Name****First Name**

Robbins

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## Inventor Name Search

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**Last Name****First Name**

Lu

Lina

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DOCUMENT TYPE: Article  
LANGUAGE: English

AB Bone marrow-derived dendritic cells (DCs) can be genetically engineered using adenoviral Ad vectors to express immunosuppressive molecules that promote T cell unresponsiveness. The numbers of these DCs for therapy of allograft rejection has been limited by partly the potential of the adenovirus to generate unwanted side effects and the limited ability of the DCs to elicit potent immune responses. We have generated a DC transduction system via NF-kappaB-dependent promoter, which can be used to generate "transgenic" DCs that express a variety of genes. We have generated DCs expressing human CD80 and CD86, or expressing the transgene of NF-kappaB inhibitor, I-kappaB kinase and CTLA4-Ig (Ad CTLA4-Ig) to generate simply immature dendritic cells that secrete the potent costimulation blocking agent. These Ad CTLA4-Ig-transduced ODN DCs exhibit markedly impaired allostimulatory ability and promote apoptosis of activated T cells. Furthermore, administration of Ad CTLA4-Ig ODN-treated donor DCs (C57BL/6; B10.H-2k) before transplant significantly prolongs MHC-mismatched (C57BL/6; B10.H-2k) vascularized heart allograft survival, with long-term (>100 days) donor-specific graft survival in 40% of recipients. The mechanism(s) responsible for DC **tolerogenicity**, which may involve activation-induced apoptosis of alloreactive T cells, do not lead to skewing of intragraft Th cytokine responses. Use of NF-kappaB antisense decoys in conjunction with rAd encoding a potent costimulation blocking agent offers promise for therapy of allograft rejection or autoimmune disease with minimization of systemic immunosuppression.

L6 ANSWER 2 OF 2 CALLING SIGNIFICANT OF 1000

ACCESSION NUMBER: 100-100000000

[illegible]

TITLE: 1. Induction of tolerogenic dendritic cells for  
2. antigen presentation  
3. tolerogenicity in a murine model  
4. of type 1 diabetes

INVENTOR(S): John, Michael; Lin, Wlannoukakis, Nick

PATENT ASSIGNMENT TO: University of Pittsburgh of the Commonwealth System of Higher Education, PA

SOURCE: FBI Int. Aff., 64 pp.

Table 1. *Salmonella* serotypes and their associated diseases

DOCUMENT TYPE: Patent.

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO.    | KIND | DATE       | ANALYST'S NAME | DATE       |
|---------------|------|------------|----------------|------------|
| WO 2001083713 | A1   | 2001-11-08 | WANG, JI-FENG  | 2001-11-09 |
| WO 2001083713 | A1   | 2001-12-14 |                |            |

|    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| W: | AM | AD | AI | AK | AL | AN | AO | AP | AR | AS | AT | AV | AW | AX | AY | AZ |
|    | A  | D  | I  | K  | L  | N  | O  | P  | R  | S  | T  | V  | W  | X  | Y  | Z  |
| BB | BB | CC | DD | EE | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ |
| CC | DD | EE | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS |
| DD | EE | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT |
| EE | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU |
| FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV |
| GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW |
| HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX |
| II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY |
| JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |

[illegible]

A5 The present invention relates to a tolerogenic mammalian dendritic cell. The said method for the preparation of the tolerogenic DCs. In addition, the present invention provides a method for enhancing tolerogenicity in a subject comprising administering the tolerogenic mammalian cell of the present invention to the host. The tolerogenic cell of the present invention comprises a library expression vector. The expression vector

NF-kappa.B signaling system. The tolerogenic role of the present invention may further comprise a third, fourth, and preferably an additional fourth, fifth, sixth, seventh, and eighth, the tolerogenicity of the tolerogenic cells and present methods. Enhanced tolerogenicity is useful for prolonging foreign graft survival and for treating inflammatory related diseases, such as autoimmune diseases.

16 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2002 ACP

ACCESSION NUMBER: 2001:922841 CAPLUS

DOCUMENT NUMBER: 137:45987

TITLE: The role of Stat5 in the induction of regulatory T cells in transplantation tolerance

AUTHOR(S): Stepkowski, S. M.; Kirken, R. A.; Hany, A. A.; Trawick, R. W.; Wang, M.; Delaney, R.; Wang, M.-P.; Han, L.; Clark, J.; Fehn, R. L.

CORPORATE SOURCE: and Department of Immunologic Biology, University of Texas, Houston, TX, USA

SOURCE: and transplantation models of 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118, 119, 120, 121, 122, 123, 124, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 138, 139, 140, 141, 142, 143, 144, 145, 146, 147, 148, 149, 150, 151, 152, 153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 172, 173, 174, 175, 176, 177, 178, 179, 180, 181, 182, 183, 184, 185, 186, 187, 188, 189, 190, 191, 192, 193, 194, 195, 196, 197, 198, 199, 200, 201, 202, 203, 204, 205, 206, 207, 208, 209, 210, 211, 212, 213, 214, 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415, 416, 417, 418, 419, 420, 421, 422, 423, 424, 425, 426, 427, 428, 429, 430, 431, 432, 433, 434, 435, 436, 437, 438, 439, 440, 441, 442, 443, 444, 445, 446, 447, 448, 449, 450, 451, 452, 453, 454, 455, 456, 457, 458, 459, 460, 461, 462, 463, 464, 465, 466, 467, 468, 469, 470, 471, 472, 473, 474, 475, 476, 477, 478, 479, 480, 481, 482, 483, 484, 485, 486, 487, 488, 489, 490, 491, 492, 493, 494, 495, 496, 497, 498, 499, 500, 501, 502, 503, 504, 505, 506, 507, 508, 509, 510, 511, 512, 513, 514, 515, 516, 517, 518, 519, 520, 521, 522, 523, 524, 525, 526, 527, 528, 529, 530, 531, 532, 533, 534, 535, 536, 537, 538, 539, 540, 541, 542, 543, 544, 545, 546, 547, 548, 549, 550, 551, 552, 553, 554, 555, 556, 557, 558, 559, 560, 561, 562, 563, 564, 565, 566, 567, 568, 569, 570, 571, 572, 573, 574, 575, 576, 577, 578, 579, 580, 581, 582, 583, 584, 585, 586, 587, 588, 589, 590, 591, 592, 593, 594, 595, 596, 597, 598, 599, 600, 601, 602, 603, 604, 605, 606, 607, 608, 609, 610, 611, 612, 613, 614, 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1178, 1179, 1180, 1181, 1182, 1183, 1184, 1185, 1186, 1187, 1188, 1189, 1190, 1191, 1192, 1193, 1194, 1195, 1196, 1197, 1198, 1199, 1200, 1201, 1202, 1203, 1204, 1205, 1206, 1207, 1208, 1209, 1210, 1211, 1212, 1213, 1214, 1215, 1216, 1217, 1218, 1219, 1220, 1221, 1222, 1223, 1224, 1225, 1226, 1227, 1228, 1229, 1230, 1231, 1232, 1233, 1234, 1235, 1236, 1237, 1238, 1239, 1240, 1241, 1242, 1243, 1244, 1245, 1246, 1247, 1248, 1249, 1250, 1251, 1252, 1253, 1254, 1255, 1256, 1257, 1258, 1259, 1260, 1261, 1262, 1263, 1264, 1265, 1266, 1267, 1268, 1269, 1270, 1271, 1272, 1273, 1274, 1275, 1276, 1277, 1278, 1279, 1280, 1281, 1282, 1283, 1284, 1285, 1286, 1287, 1288, 1289, 1290, 1291, 1292, 1293, 1294, 1295, 1296, 1297, 1298, 1299, 1300, 1301, 1302, 1303, 1304, 1305, 1306, 1307, 1308, 1309, 1310, 1311, 1312, 1313, 1314, 1315, 1316, 1317, 1318, 1319, 1320, 1321, 1322, 1323, 1324, 1325, 1326, 1327, 1328, 1329, 1330, 1331, 1332, 1333, 1334, 1335, 1336, 1337, 1338, 1339, 1340, 1341, 1342, 1343, 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16 ANSWER = 100%  
ACCESSION NUMBER: 141410  
DOCUMENT NUMBER: 141410  
TITLE: Transplantation of murine allograft survival using  
depleted cells treated with NF-kappa.B decoy  
oligonucleotides  
AUTHOR S : Hannekhan, Nick; Bonham, C. Andrew; Qian, Shiguang;  
Shou, Zhengyou; Peng, Liansha; Harnaha, Jo; Li, Wei;  
Thomson, Angus W.; Fung, John J.; Robbins, Paul D.;  
Lu, Lina  
CORPORATE SOURCE: Department of Molecular Genetics and Biochemistry,  
University of Pittsburgh, Pittsburgh, PA, 15261, USA  
SOURCE: Molecular Therapy (2005), 15, Pt. 1, 4: 422  
CODEN: MTHCHK; ISSN: 1525-0167  
PUBLISHER: Academic Press  
DOCUMENT TYPE: Journal

12 ANNOTATED BIBLIOGRAPHY MEDICAL ABSTRACTS INTERNATIONAL  
ACCESSION NUMBER: 1991-12-1000  
DOCUMENT NUMBER: 1991-12-1000  
TITLE: Manipulation of **dendritic cells** for tolerance induction in transplantation and autoimmune disease.

AUTHOR(S): Lu, Lin L.; Thomas, Anne W.  
CORPORATE SOURCE: Dr. Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, 350 Lothrop Street, E1554, Pittsburgh Science Tower, Pittsburgh, PA, 15261; LuL@msx.upmc.edu USA  
SOURCE: Transplantation (Baltimore), (January 15, 2002) Vol. 73, No. 1 Supplement, pp. S16-S21.  
<http://www.transplantationjournal.com/print>.  
ISSN: 0041-1987.

DOCUMENT TYPE: General Review  
LANGUAGE: English

AB **Dendritic cells** (DC) have been shown to initiate and regulate immune responses. By studying DC biology, we have improved our understanding of DC biology, including their origin, development, and function. DC exist as distinct subsets that differ in their lineage affiliation, surface molecule expression, and biological function. These factors seem to determine the T-cell polarizing signals and type of T cell response-T helper 1, T helper 2, or T regulatory-induced by DC (1). Evidence has accumulated that DC play an important role in both central and peripheral tolerance via various mechanisms, including induction of T-cell anergy, immune deviation, T regulatory cell activity, and promotion of activated T-cell apoptosis. Although many of the details of the molecular basis of DC **tolerogenicity** have yet to be elucidated, emerging information suggests that costimulatory molecule deficiency, expression of death-inducing ligands (in particular Fas (CD95) ligand), microenvironmental factors (in particular anti-inflammatory/immunosuppressive cytokines), and inhibition of gene transcription regulatory proteins (e.g., nuclear factor-kappaB) can impact **tolerogenic** potential of DC (2). Manipulation of DC by control of their maturation and differentiation, or genetic engineering of these cells to express immunosuppressive molecules, shows potential for therapy of all solid organ and hematopoietic diseases. In this review, we discuss the principles and techniques for generation of "tolerogenic" DC and summarize the data from experimental models. These concepts may be applicable to clinical trials.

13 ANNOTATED BIBLIOGRAPHY MEDICAL ABSTRACTS INTERNATIONAL  
ACCESSION NUMBER: 1991-12-1000  
DOCUMENT NUMBER: 1991-12-1000  
TITLE: Modulation of cellular allograft survival by **dendritic cells** genetically engineered with NF-kappaB oligodeoxynucleotide decoys and dendritic proteins such as CD137L.  
AUTHOR(S): Benker, J. Andrew; Lu, Lin L.; Thomas, Anne W.; Chen, Tony W.; Wang, Liang; Ma, Lili; Barkstein, Hilary; Robbins, Paul D.; Thomas, Anne W.; Fung, John C.; Lu, Lin L.; Chinn, J.  
CORPORATE SOURCE: Dr. Thomas E. Starzl Transplantation Institute, University of Pittsburgh Medical Center, 350 Lothrop Street, Pittsburgh Science Tower, E1554, Pittsburgh, PA, 15261; LuL@msx.upmc.edu USA  
SOURCE: Transplantation (Baltimore), (January 15, 2002) Vol. 73, No. 1 Supplement, pp. S22-S27.  
<http://www.transplantationjournal.com/print>.

14 ANNOTATED BIBLIOGRAPHY  
LANGUAGE: English

AB **Dendritic cells** - **dendritic cells** - **dendritic cells**

18 ANSWER : YES  
ACCESS : 11/11/2004  
DOCUMENT NUMBER :  
TITLE : The use of tolerogenic dendritic cells for enhancing tolerogenicity in a host and methods for making the same  
INVENTOR(S) : Kourlis, Paul D.; Lu, Ming; Giannoukakis, Nick  
PATENT ASSIGNMENT : University of Pittsburgh of the Commonwealth System of Higher Education, USA  
SOURCE : PCT Int. Appl., 63 pp.  
OPEN : BAKING  
DOCUMENT TYPE : Patent  
LANGUAGE : English  
FAMILY ACC. NUM. COUNT : 1  
PATENT INFORMATION :

| PATENT NO.     |    | BIRTH DATE  |    |    |    |    |    | APPLICANT'S NAME  |    |    |    |    |    |    |    |    |    | GATE  |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
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| WO 2001/012345 |    | A. B. C. D. E. F. G. H. I. J. K. L. M. N. O. P. Q. R. S. T. U. V. W. X. Y. Z. |    |    |    |    |    | A. B. C. D. E. F. G. H. I. J. K. L. M. N. O. P. Q. R. S. T. U. V. W. X. Y. Z. |    |    |    |    |    |    |    |    |    | A. B. C. D. E. F. G. H. I. J. K. L. M. N. O. P. Q. R. S. T. U. V. W. X. Y. Z. |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| NO:            | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
| NO:            | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
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|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
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|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
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|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
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|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
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|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
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|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
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|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX | YZ | AA | BB | CC | DD | EE  | FF | GG | HH | II | JJ | KK | LL | MM | NN | OO | PP | QQ | RR | SS | TT | UU | VV | WW | XX | YY | ZZ |
|                | AB | CD  | EF | GH | IJ | KL | MN | OP  | QR | ST | UV | WX |    |    |    |    |    |   |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |

The present invention relates to a tolerogenic mammalian dendritic cells ("DCs") and methods for the preparation of the tolerogenic DCs. In addition, the present invention provides a method for enhancing tolerogenicity in a host comprising administering the tolerogenic mammalian DCs to the patient in accordance to the claim. The tolerogenic DCs of the present invention comprise an IL-6 signal transducer (IL-6R) gene, NF-kappaB inhibitor, and the tolerogenic DCs of the present invention may further comprise a costimulatory molecule such as B7-1 or B7-2 and CD80 or CD86.

AB The present invention relates to a tolerogenic mammalian dendritic cells ("DCs") and methods for the preparation of the tolerogenic DCs. In addition, the present invention provides a method for enhancing tolerogenicity in a host comprising administering the tolerogenic mammalian DCs to the patient in accordance to the claim. The tolerogenic DCs of the present invention comprise an IL-6 signal transducer (IL-6R) gene, NF-kappaB inhibitor, and the tolerogenic DCs of the present invention may further comprise a costimulatory molecule such as B7-1 or B7-2 and CD80 or CD86.

the tolerogenic DCs when present. Therefore, enhanced tolerogenicity in a host is useful for promoting tumor cell survival and for treating inflammatory related diseases, such as autoimmune diseases.

LE ANSWER # 00000000000000000000  
ACCESSION NUMBER: 00000000000000000000  
DOCUMENT NUMBER: 00000000000000000000  
TITLE: Tolerogenic dendritic cells  
AUTHOR(S): Bressanini, M.; Bianchi, E.; Hsi, R.;  
Takahashi, S.; D'Alella, C.; Dalla-Favera, R.;  
Bressanini, E.  
CORPORATE SOURCE: Department of Pathology, Columbia University, New  
York, NY, USA  
SOURCE: Human Immunology 62(1), 62(10), 1988-1972  
CODEN: HUMIMH; ISSN: 0198-8950  
PUBLISHER: Elsevier Science Inc.  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Dendritic cells are crucial to the activation as well as suppression of the immune response. Previous reports have illustrated that APC interacting with antigen-specific T suppressor cells become **tolerogenic**, inducing T helper anergy. To characterize the changes occurring in **tolerogenic APC**, the mRNA profile of K561 **dendritic cells** exposed to allo-specific T helper and T suppressor cells was analyzed. Our study will provide evidence that immature dendritic cells can induce T suppressor cells and that mature dendritic cells with a distinct phenotype are involved in the regulatory pathways of dendritic cell differentiation and development. We intend to use these strategies.

REFERENCE COUNT: 47 THESE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

LE ANSWER 5 OF 6 CAPTION: COPYRIGHT 2012 APS  
ACCESSION NUMBER: 1991199123 CAPTION  
DOCUMENT NUMBER: 1991199123  
TITLE: Prolongation of cardiac allograft survival using  
dendritic cells treated with  
NF-kappa.B decoy oligodeoxynucleotides  
AUTHOR(S): Giannoukakis, Nikk; Berham, C. Andrew; Jian, Shizhen;  
Zhou, Zhongyou; Peng, Lianshe; Barnaba, Jo; Li, Wei;  
Therrien, Anne M.; Peng, Jun J.; Perkins, David L;  
Li, Lina  
CORPORATE SOURCE: Department of Molecular Genetics and Biotechnology,  
University of Pittsburgh, Pittsburgh, PA, 15261, USA  
SOURCE: \*JOURNAL OF CLINICAL INVESTIGATION\* 117(12):3041-3050, 2007  
PUBLISHED: 2007  
DOCUMENT DATE: 2007  
LANGUAGE: English

AB Dendritic cells (DCs) are highly specialized cells that play a central role in the immune system. They are found in all tissues and are responsible for capturing, processing, and presenting antigens to T cells. The expression of co-stimulatory molecules, such as B7-1, B7-2, and ICAM-1, is crucial for DC function. In this study, we investigated the expression of these molecules in DCs from tolerant and non-tolerant mice. We found that tolerant DCs express significantly lower levels of B7-1 and B7-2 compared to non-tolerant DCs. This suggests that the expression of co-stimulatory molecules is a key factor in the development of tolerance. Furthermore, we observed that tolerant DCs have a higher capacity to induce regulatory T cells (Tregs), which are known to suppress immune responses. These findings provide insight into the mechanisms of tolerance and may have implications for the treatment of autoimmune diseases.

transcription of a reported gene. However, exposure of DC to the oxidant, hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>), significantly suppressed nitric oxide synthase activity and proliferation. Treatment of bone marrow-derived DC pretreated with NF- $\kappa$ B with H<sub>2</sub>O<sub>2</sub> significantly suppressed the cell-surface expression of costimulatory molecules without interfering with MHC class II or class II expression. Furthermore, NF- $\kappa$ B ODN DC induced allogeneic donor-specific hyporesponsiveness in mixed leukocyte cultures, and this was associated with inhibition of Th1-type cytokine production. Finally, infusion of NF- $\kappa$ B ODN-modified bone marrow-derived DC into allogeneic recipients prior to heart transplantation resulted in significant prolongation of allograft survival in the absence of immunosuppression. Specific interference with NF- $\kappa$ B and other transcriptional pathways involved in immune stimulation in DC using decoy approaches could be the means to promote tolerance and prevent organ transplantation. (Y. Li, J. Anagnostou, E. J. Wherry)

REFERENCE COUNT: 24 THREE ARE CITED REFERENCES AVAILABLE FOR THIS SPECIAL. ALL CITATIONS AVAILABLE IN THE CD FORMAT